The Journal

of the

Louisiana State Medical Society

$4.00 Per Annum, 35c Per Copy
Vol. 113, No. 10

OCTOBER, 1961

Published Monthly
1430 Tulane Avenue, New Orleans 12, La.

Injuries of the Urinary Tract*

• The treatment of these injuries together with the medico-legal aspects of the problem are gone into carefully and thoroughly.

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Introduction

MANAGEMENT of the patient with urinary tract injury aims to restablish an intact urinary tract, control hemorrhage and to conserve the involved urinary organs. To achieve this goal, accurate diagnosis must precede definitive therapy.

Despite the protection afforded the kidney by its deep position and shelter of surrounding structures, it is often injured by various forms of trauma that leave neighboring organs and structures unharmed. Approximately four-fifths of kidney injuries occur without injury to other structures. However, in the lower urinary tract, particularly in the male, the problem is often urogenital since the two systems are closely integrated.

Automobile collisions, industrial and sport accidents account for the great majority of wounds. Approximately twenty per cent of injuries occur in children. Bicycles, velocipedes, scooters and skates account for a goodly number of childhood casualties. One of my former residents in urology lost a kidney as a result of a football injury sustained in a high-school football game. Penetrating missiles or sharp objects may damage the kidney. In a personal case a revolver bullet necessitated nephrectomy and, strangely, no other viscus was involved. Statistics from the South show a high percentage of knife wounds, while in the North the automobile is chief offender.

Medico-Legal Aspects

A very important phase of injuries to the urinary tract, *and this applies to all injuries*, are their medico-legal and industrial aspects. We are inclined to neglect this most important aspect of injuries in our single-minded effort to treat the patient and get him well. It is my firm conviction that medical schools should provide instruction that familiarizes students with at least the general principles of court procedure and the real or potential medico-legal problems that are bound to confront them in actual practice.

Much as we may dislike this phase of medical practice, we should expect and be prepared, as a specialist or general practitioner to testify in court, submit reports, or render opinions concerning the

* Presented at the Twenty-fourth Annual Meeting of the New Orleans Graduate Assembly, March 6-9, 1961, New Orleans.

diagnosis and treatment in cases of industrial or accidental injuries. The doctor's appearance in court is not only an obligation, but essential to the administration of justice and a correct legal decision. He should be able to uphold the dignity of the medical profession by knowing how to conduct himself on the witness stand, thereby avoiding embarrassment and humiliation at the hands of clever cross-examiners who are too often interested only in confusing the issue, with frequent miscarriage of justice.

Because of the possible medico-legal aspects of trauma of any sort, it is most important to record in minute detail an accurate history of the injury and the results of a meticulous examination.

Injuries of the Kidney

It is well to remember that relatively light trauma may injure a kidney with silent, pre-existing disease, such as hydronephrosis, renal calculi, cysts, tumors or chronic infection. I emphasize the word *silent* because many different diseases of the kidney can insidiously lead to its destruction without the patient's knowledge that anything is wrong. In court the medical expert must be alert to forestall attempts of the claimant to attribute the pre-existing disease to the alleged injury. It has been my misfortune to be involved in several cases of this kind. For example, a freight car conductor was thrown off balance when the engineer stopped the train rather suddenly. He complained of some injury to his back, but on examination no evidence of injury could be found. He had no hematuria, not even microscopic hematuria. One of the most important rules to remember is that injury to the kidney, even though a minimal contusion, almost invariably produces red blood cells in the urine. This man had none. However, on urological investigation he was found to have a left kidney destroyed by an old calculous pyelonephritis in no way related to the recent injury. As often happens in a jury trial when the plaintiff appears as an individual against a corporation (in this case the railroad) he won the jury's sympathy and an award of $37,500.00.

The kidney is the most vascular organ in the body and therefore vulnerable to profuse hemorrhage manifesting itself in the urine and in the perirenal tissues. The kidney may be likened to a ball of fluid and the impact of a blow is transmitted in all directions within the organ. Sargent and Marquardt classify renal injuries with respect to severity into three groups:

A. **Contused Kidney** (58 per cent). The injury consists of superficial laceration, minimal shock and pain, no mass in the loin, hematuria microscopic or macroscopic, normal perirenal shadows by x-ray and almost intact pyelogram.

B. **Fractured Kidney** (35 per cent). Greater degree of shock, pain in the loin, muscular rigidity, possibly a palpable mass in the loin (due to extravasation of blood or urine or both), hematuria (gross). Pyelograms may show opaque medium passing from pelvis into parenchyma or through the renal capsule into the perirenal tissue. The perirenal fascia will confine the extravasation to the area immediately surrounding the kidney.

C. **Fragmentation of Kidney** (9 to 17 per cent). The clinical picture reflects massive blood loss with urinary extravasation, producing a rapidly increasing mass in the flank and profound shock. Fragmentation of the kidney may include tearing of major vessels or complete disruption of the renal pedicle. Such cases demand prompt surgical intervention as a life-saving measure without the opportunity for preliminary study.

The contused kidney (A) will heal with conservative treatment. In the fractured kidney (B) non-surgical management is preferable until a definite indication for surgical intervention is observed. Urine does not extravasate from the torn kidney surface unless a calyx or the renal pelvis is involved. It is important with respect to treatment to remember that a certain amount of hematoma and even extravasated urine can be reabsorbed without residual damage. Unlike ruptures of the

liver or spleen, bleeding about the kidney tends to be contained to a degree by the surrounding capsule which fortunately exerts a tamponade effect.

The most important immediate diagnostic procedure following initial clinical evaluation and treatment of shock is x-ray examination. A routine plain K.U.B. (kidneys-ureters-bladder scout film of the abdomen) followed by intravenous urography should be carried out. Retrograde pyelography may be done (patient's condition permitting) if intravenous studies are not sufficiently revealing. Complete bed rest is mandatory. Usually gross hematuria will subside in a period of two or three days but may keep up for as long as two weeks.

Conservative treatment is advocated because a hasty operation often means a hasty nephrectomy in cases where the kidney might have been saved by judicious waiting. Personal experience indicates that about eighty per cent of cases can be handled by conservative treatment. The pressure of increasing renal tumefaction, unrelenting hematuria, x-ray or clinical evidence of extravasation of urine or severe persistent pain is indication for surgical exploration. An important sign of increasing hematoma or urinary extravasation is severe pain radiating down to the groin or testicle. It is of course necessary to know the condition of the opposite kidney and to evaluate its functional capacity in dealing surgically with the injured kidney, and also the presence or absence of injury to other organs.

Regardless of whether or not the injured kidney is treated surgically it should be re-evaluated in two or three months, and again in six months by intravenous urography because of the possibility that scar tissue obstruction of the ureter might cause hydronephrosis, unresolved hematomas which may become calcified, and secondary pyelonephritis.

Injuries of the Ureter

Due to its small caliber and deep position the ureter is not often involved in the type of injuries which so often affect the kidney. Trauma to the ureter is most often sustained as a result of surgical injuries incidental to operations on the pelvic organs, especially hysterectomy. Resection of the large bowel for neoplasms may likewise result in ligation or section of the ureter. It is always well to precede pelvic surgery with intravenous urography, and more frequent use of indwelling ureteral catheters during surgery is heartily recommended. The ureters are in close juxtaposition to the cervix and uterine arteries, rendering them extremely vulnerable to surgical accidents. I have seen instances in which inadvertent ligation of a ureter during pelvic surgery has gone unrecognized because it did not produce appreciable immediate symptoms. The ligated ureter does not cause renal atrophy. On the contrary, hydronephrosis develops which may years later cause symptoms necessitating nephrectomy. In one personal case the hysterectomy was done two years earlier, and in another twelve years. In the latter case the kidney was a thin walled sac containing over 1000 cc. of fluid. Accidental obstruction of both ureters results in anuria and demands prompt surgical intervention as a life saving measure.

Injuries of the Bladder

In civil life rupture is the usual consequence of injury to the bladder, whereas in war perforation by missiles is the usual injury. Ruptures may be intra- or extraperitoneal. Injuries causing fracture of the pelvis are most often associated with extraperitoneal rupture. If distended, the bladder can be easily ruptured by an external blow or even sudden pressure. This is especially true of the hypalgesic bladder of the alcoholic or tabetic person. The clinical evidence of rupture is swelling and rigidity of the lower abdomen and inability to void together with pain and shock. Catheterization often yields urine which is not at all blood-stained.

The most important diagnostic measures are intravenous urography and retrograde cystography. The latter procedure is more effective in revealing extravasa-

tion. The urethra should not be overlooked, because the prostatic and bulbomembranous portions of the urethra are prone to injury as a result of their attachments under the pubic arch. It is not uncommon to see the prostatic urethra completely avulsed and torn away from the bulbomembranous urethra in crushing injuries of the pelvis, leaving the bladder and prostate floating free in the pelvis in a pool of blood and urine. I have not found this such a formidable problem to handle, because it is not difficult to do a cystotomy and re-establish the continuity of the urethra with a Foley catheter using gentle traction to bring the prostate back to its normal position. The urethra will coapt and heal without the use of sutures if the catheter is maintained in place for ten days to two weeks. In rupture of the bladder it is interesting to note that clean urine escaping into the peritoneal cavity does not irritate the peritoneum and peritonitis will not occur unless the urine is infected. In a personal case the patient under the influence of alcohol fell over the foot of his bed and ruptured an overdistended bladder. At operation a loop of ileum was found protruding into the bladder through a rent in its posterior wall. Four thousand cubic centimeters of urine were aspirated from the peritoneal cavity. The latter revealed no evidence of inflammatory reaction, and the patient made a prompt recovery.

Injuries of the Urethra

Injuries to the urethra are of serious import because of stricture formation, requiring treatment (in many instances) for the rest of the patient's life.

Injuries involving fracture of the pelvis, as I have already mentioned, are frequently associated with rupture of the bladder, but perhaps more frequently with rupture of the urethra. Both lesions may occur in the same patient. Fractures of the pelvis most often cause rupture of the urethra proximal to the urogenital diaphragm, with intrapelvic extravasation. The most common urethral injuries in my experience have been the so-called "straddle injuries", caused by falling astride some hard object so that sudden force is exerted against the perineum, compressing the bulbomembranous urethra against the pubic arch. Hemorrhage and urinary extravasation in these cases is distal to the urogenital diaphragm and extends within the confines of Colle's fascia and eventually beneath Scarpa's fascia of the abdomen. These injuries differ from others in the urinary tract by the absence of immediate urinary extravasation. Urine escapes into the tissues only when the patient attempts to void and finds that he cannot do so. Blood only, will ooze from the urethra, and efforts at catheterization usually fail because of loss of continuity of the urethral mucosa. Such efforts usually provoke fresh hemorrhage as well as "urethral chills" as a result of bacteremic shock unless pursued with meticulous attention to asepsis and gentle manipulation. It is most important to cleanse the external area with Phisohex or Septisol and to irrigate the anterior urethra with some antiseptic solution such as 1-10,000 zephiran before attempting to pass a catheter. If the catheter does not pass easily no force should be used. Forcible manipulation may be lethal. It is far safer to do a cystotomy and then to re-establish the continuity of the urethra by an antegrade procedure, immediately, if the patient's general condition permits or at a later date if other considerations prevent its immediate accomplishment.

The surgical technic of restoring the continuity of the urethra is comparatively simple in the hands of someone experienced in this field. The Davis interlocking sounds are of great help but not absolutely essential. I prefer to have the patient in lithotomy position so as to have access to both the perineum and the suprapubic area. The female sound is first passed down through the cystotomy wound into the prostatic urethra until its tip can be palpated in the perineum. The male sound is then passed into the external urethral meatus and anterior urethra

until the two sounds come together. The male sound then follows the female sound on into the bladder. It is then a simple matter to attach a catheter and draw it through the entire urethra where it is left indwelling for about two weeks, by which time the continuity of the urethra will be re-established. An alternate procedure, which is easily carried out, is to pass a coudé catheter or a metal catheter through the cystotomy wound and incise the perineum directly over the tip of the catheter when it is palpated in the perineum. A Foley catheter, preferably 24 or 26 F, passed by way of the anterior urethra down to the perineal incision, is attached to the other catheter and drawn into the bladder. I believe that the perineal incision is in general, an advantage, as it permits better drainage of the ruptured area and therefore reduces the likelihood of stricture formation. No sutures are necessary to approximate the *torn urethral ends*. The indwelling catheter serves as a scaffolding or splint favoring prompt union of the severed ends of the urethra.

I have used on many occasions this same technic for the treatment of impassable strictures of the urethra resulting from old gonorrheal infections. In many instances these patients were first seen in extremis with periurethral abscesses and urinary extravasation with multiple fistulae. In these cases, the stricture is usually in the bulbomembranous urethra. The excess scar tissue is excised through the perineal incision, the continuity of the urethra is re-constituted with a Foley catheter without any attempt to suture the ends of the urethra together, and the results have been most gratifying. Morbidity and mortality have been reduced to the vanishing point with this simple course of action. Follow-up care is important. Sounds should be passed periodically until one is assured that the danger of further stricture formation is past.

Straddle injuries do not always rupture the urethra. In some instances the injury to the urethra is slight, and the patient is still able to void. The soreness soon departs and he develops a false sense of security until several months have passed. Then he begins to notice diminution in the size and force of his stream and eventually becomes unable to void at all. The realization is then brought home to him that he has developed a traumatic stricture as a result of straddle injury. The traumatic stricture thus formed is usually even more rigid and resistant to dilatation than the gonorrheal stricture.

In conclusion we see that there are three separate entities:

1. Rupture of the urethra.
2. Impassable gonorrheal stricture of the urethra.
3. Straddle injuries with either rupture of the urethra or delayed stricture formation.

The treatment consisting of re-establishment of the normal continuity of the urethra is the same for all three, as outlined above.

Injuries of the External Genitalia

Trauma to the penis and testicles of an industrial nature is relatively uncommon in spite of their exposed position. War wounds differ from injuries in civil life in that they are more often penetrating wounds due to bullets and shell fragments. A favorite diabolical instrument of the enemy was the mine which, when stepped on, often succeeded in blowing off the entire genitalia. In civilian life I have seen a few cases in which the integuments of the genitalia were avulsed, and in some cases partial or total emasculation accomplished. One instance a farmer, age 30, while leaning over his tractor engine, caught the front of his trousers in the fan. A remarkably clean job of emasculation was done except for one testicle which was left hanging free without any covering. The testicle was placed under the skin on the inner surface of his thigh. His urinary meatus is now in the perineum just in front of the anus.

Another young man, age 24, while using a rotary drill, had the handle catch in his trousers resulting in instant castration. On examination his scrotum was found to be intact except for a transverse

slit which looked as if it had been done with a scalpel. The stumps of the spermatic cords did not bleed because of the rotary effect of the drill. All we had to do was to suture the scrotum and put the young man on testosterone treatment to control his hot flashes.

The question of traumatic epididymitis and epididymo-orchitis has given rise to considerable acrimonious debate in the field of industrial medicine. It is probable that the insurance carrier has often been made to pay compensation for instances of alleged injury which were in reality cases of acute nonspecific epididymitis unrelated to injury, or at least to any direct trauma to the organ.

In conclusion, I hope that this discursive account of injuries to the urogenital organs may have been the source of a few practical gleanings for the audience for whom it was intended.

Virchow on Soups and Broths

The distinguished German professor and politician has been accused of being the chief opponent of soup. He says that this is not true, for he had merely said that meat broths are neither nutritious nor "substantial." That if all the meat which one uses should be boiled and soup made of it, the meat would become for the greater part indigestible, and the soup would not be a substitute for it. Ordinary meat broth or bouillon in its pure form can only be recognized as a condiment. By the addition of eggs, flour, fat and other things, it may acquire a certain nourishing and heating value. * * * Taken warm, it is of nearly the same value as coffee or tea, but is inferior to wine, schnapps, or beer; it only stimulates the nerves. It has one advantage over every other condiment, namely, it contains no poisonous substance, it is incomparably milder, hence much better adapted to feeble persons; and finally, it can be conveniently combined with substances that are actually nutritious, and imparts to them an agreeable and "substantial" taste.—**Scientific American,** April 8, 1882 **New England Medical Monthly.**

New Orleans M. & S. J. 10:295 (Oct.) 1882.

Laboratory Diagnosis of Diabetes Mellitus*

• Laboratory diagnosis of diabetes is made more inclusive by determination of postprandial blood sugars.

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CARBOHYDRATE is the chief metabolite of the body process. Regular ingestion is necessary as the body stores are limited in a 70 kilogram person to 300 gms. or about one half a day's energy requirements. If this is not maintained, then proteins and fats are metabolized to supply this energy.

Chemical Processes

Polysaccharides and disaccharides are hydrolyzed in the gastrointestinal tract to monosaccharides and are apparently absorbed by either diffusion or phosphorylation. Absorption rate varies with each saccharide and is influenced by other food in the tract, physiological state of the intestinal mucosa, emptying time of the stomach and endocrine activity. The latter is the activity of the thyroid and adrenal cortex and is not influenced by insulin activity.

Glucose, a hexose, is the only carbohydrate found in the blood of a normal fasting person. It is stored as glycogen, a polymer. Glycogen is present only in the intracellular component and represents 70 per cent of the carbohydrate total. In contrast, glucose is found only in extracellular fluids.

As glucose enters the cell membrane it is phosphorylated and can be metabolized. The enzyme-catalyze reaction can convert glucose into glycogen, oxydize it to CO_2 and water with energy release, or convert it by two carbon fragment degradation into fat or amino acids.

Homeostatic Factors Involve All Tissues

A number of delicately balanced homeostatic factors maintain the blood glucose level to a narrow range. This level is constant throughout life and only infants born of either normal or diabetic mothers exhibit a transient hypoglycemia during the first twelve to thirty-six hours after birth. The homeostatic factors include, hepatic and renal uptake and release of glucose, peripheral tissue removal and hormonal influence while gastrointestinal absorption has only a temporary effect upon the level.

The liver and kidney are the only endogenous sources of blood sugar. The liver is the most important and can remove or contribute glucose to the blood or synthesize it from a number of amino acids and fatty acid fragments. The mechanism is not clear, but under certain circumstances the kidney can release glucose into the circulation.

Glucagon and Other Hormones Influenced by Sugar Levels

The blood sugar level serves as a stimulus to the liver in determining whether glycogenesis or glycogenolysis shall predominate. Hyperglycemia in the presence of normal amounts of insulin inhibits the hepatic output of glucose while accelerating its deposition. Both of these processes aid in restoring the blood glucose to normal. Hypoglycemia is associated with decreased hepatic gluconeogenesis and with increased glucose output. The thyroid, adrenal cortex and anterior pituitary gland influence blood sugar levels by hormonal effect on the liver. On the other hand, insulin seems to act on the extra-

* Presented at the Eighty-first Annual Meeting of the Louisiana State Medical Society, May 9, 1961, in New Orleans.

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hepatic tissue. Glucagon is a hormone of the alpha cells of the pancreas and increases the blood glucose level by a glycogenolytic effect on liver glycogen. In emergencies, epinephrine can cause excessive hepatic glycogenolysis and hyperglycemia.

All peripheral tissues utilize glucose for energy purposes and of these only cardiac or skeletal muscle can store a significant amount as glycogen. There is a direct relationship between blood glucose concentration and utilization of carbohydrate by extrahepatic tissues. This holds true for both normal and diabetic patients; although the rate of utilization is lower in the diabetic group at a given glucose concentration. In contrast, when hypoglycemia is present, the rate of entry of glucose into muscle will vary with the cause of the decreased blood sugar. Insulin is the one hormone which has been shown to increase the extrahepatic uptake of glucose.

Abnormal blood sugar concentrations result from an imbalance between hepatic output of glucose, peripheral uptake of glucose, or disturbance in the endocrine regulatory influence upon these processes.

Hyperglycemia

Hyperglycemia may result from (1) an increase in hepatic production and release of glucose with normal removal rate by peripheral tissues, (2) a normal hepatic output of glucose with a subnormal rate of peripheral removal, or (3) a combination of these factors.

Diabetes mellitus is a syndrome characterized by persistent hyperglycemia and glycosuria. Mild diabetes may show normal fasting blood glucose levels. It may be necessary to determine a postprandial blood sugar level or do a glucose tolerance test in order to establish a diagnosis, especially since glycosuria may be absent. An overproduction of glucose by the liver is the primary abnormality in the syndrome. Glycosuria occurs when the renal tubular reabsorption capacity for glucose is exceeded. Diabetes mellitus syndrome may be caused by lesions in the islets of Langerhans or by disorders of other endocrine glands. Increased activity of the adrenal cortex, either primary or as a result of pituitary basophilism with increased ACTH production produces hyperglycemia by increasing hepatic glyconeogenesis greater than increase in peripheral uptake. Pituitary eosinophilism with increased growth hormone produces acromegaly and hyperglycemia. The mechanism is not definitely known but it may be by growth hormone influence on glyconeogenesis as well as an anti-insulin effect by glucagon production. Hyperthyroidism increases blood sugar levels if the peripheral utilization does not keep pace with the augmented hepatic output. Hyperglycemia may also be found in cases of pheochromocytoma.

Fasting Blood Sugar Levels are of Limited Use

In the diabetic syndrome, insulin restores hepatic glyconeogenesis to normal and facilitates peripheral utilization at normal blood sugar levels. Other conditions that produce increased blood sugar levels are (1) anoxia, (2) general anesthesia, (3) convulsive episodes of tetany, epilepsy, eclampsia and intracranial trauma, (4) chronic hepatic dysfunction and (5) gastrectomy with "dumping syndrome".

There is no need of estimating the level of serum glucose in the fasting state except where (1) hypoglycemia is suspected or (2) where it is desired to ascertain the effect and duration of insulin action in diabetes under therapy.

If one is tempted to screen out individuals with disorders of carbohydrate metabolism leading to hypoglycemia, then a fasting blood serum glucose determination will establish this fact, especially if combined with exercise. The disease processes associated with hyperglycemia can be more readily demonstrated when tested under the load of a meal. A postprandial blood glucose determination is then the method of choice and not a fasting blood level in screening for the presence of diabetes.

Accurate Blood Sugar Method is Available and Necessary

All practical methods require the preparation of a protein free filtrate for determining the glucose level. Most laboratories use tungstic acid for their precipitant but its use should be discontinued and either $Zn(OH)_2$ or $CuWO_4$ substituted. Tungstic acid will not precipitate the nonglucose substances in the erythrocytes and plasma like glutathione, uric acid and ergothioneine so the levels obtained and ordinarily used as normals (80 to 120 mg. per 100 ml.) include these non-glucose reducing substances. With the use of better precipitating agents true serum glucose is 70 to 105 mg. per 100 ml. and whole blood level is 60 to 90 mg. per 100 ml.

Postprandial Glucose Determination is Desirable

The ability of an individual to regulate his blood sugar can best be studied by determining blood glucose concentration after a meal or after oral or intravenous glucose. The postprandial blood glucose determination requires only one specimen one to two hours after a meal and only the degree of hyperglycemia is of importance. The glucose tolerance test can be given orally or intravenously. The blood glucose level is determined at specified time intervals before and after the oral or intravenous administration of a given amount of glucose. In the oral test, both the degree and duration of hyperglycemia are of clinical importance, whereas in the intravenous method, only the duration of hyperglycemia is significant.

Regardless of the method of administration the liver is the principal organ involved in the regulation of blood sugar and determines the response to the load of carbohydrate administered.

Oral Glucose Tolerance Test

Orally, glucose is absorbed at a rate which at first is greater than the capacity of the liver and peripheral tissues to remove it. The height of the blood sugar usually does not exceed 150 mg. per 100 ml. and this maximum is seen one-half to one hour after glucose ingestion. As a result of adjustments, the blood sugar is restored to normal after a time, although the intestinal absorption may be continuing at a maximum rate. After the initial increase in blood sugar the fall is delayed and does not return to control level for three or more hours. Hence both the height of the blood sugar response and its duration are of importance. Although it is assumed that there is a "normal" absorption of glucose from the intestine, even in normal individuals this rate is somewhat variable. Except in outspoken cases of gastrointestinal pathology, however, the postprandial serum glucose concentration will not be greatly affected by absorption rates since only the degree of hyperglycemia is important. On the other hand, the variability in absorption will affect the real glucose tolerance since both degree and duration of hyperglycemia are considered diagnostic. An abnormal degree of hyperglycemia may occur after gastrectomy since the stomach empties rapidly ("dumping syndrome"). This complication is avoided by using the intravenous test. In disease of the gastrointestinal tract associated with interference in absorption (sprue, celiac disease, enteritis and colitis) little if any hyperglycemia may be seen. Only a small increase in blood sugar value will be seen in untreated anterior pituitary and thyroid insufficiency. Patients with Addison's disease may show a normal type of curve if replacement therapy with electrolyte and water regulating hormones is adequate.

Intravenous Glucose Tolerance Test

In the intravenous test, glucose is introduced directly into the blood stream and all the variable factors of absorption are eliminated. After a fasting blood sugar is obtained ⅓ gm. of glucose per kg. of body weight as a 50 per cent glucose solution is injected intravenously over a three to five minute period (Soskin and Levine). Samples are taken at one-half, one, two and three hours. The concentration of blood glucose reaches a peak im-

mediately and then falls progressively. The maximum height has no significance but the duration of hyperglycemia is clinically important. The serum glucose normally will return to normal within one hour and may continue to fall for another hour to a level 5 to 15 mg. per 100 ml. below this. Following this dip, the level returns to approximately its normal level.

This "hypoglycemic" phase is produced by a lag in the liver's ability to change from its glycogenic activity to its role of glycogenolysis. Three types of abnormal responses after the ⅓ gm. per Kg. dosage is used can occur. These are: (1) A delay of three hours or more before the blood glucose returns to normal level. This is typical of the diabetic syndrome. The individual with mild diabetes may have a normal or only slightly elevated fasting blood sugar but will not be capable of restoring the blood sugar to the control value until three or more hours afterwards. This delay is caused by an absolute or relative insulin lack which affects hepatic homeostasis. (2) A return to normal levels between one and two hours after the injection. Some cases of liver dysfunction will produce this. A delay in the fall of the blood sugar is also found here but it differs from the response seen in diabetes in that the blood sugar returns to the original value between one and two hours. Here the abnormally functioning liver can not respond in the normal way to the stimulus of hyperglycemia and endocrine regulation. Thus, it is possible to differentiate patients with mild diabetes from those with liver dysfunction by means of the intravenous tolerance test. The oral test is incapable of making this differentiation. (3) An exaggerated hypoglycemic phase. As intravenous glucose subjects the regulatory mechanism to a marked stress resulting from the great hyperglycemia produced, the test tends to exaggerate the hypoglycemic phase as found in hyperinsulinism, glycogen storage disease and adrenal cortical and anterior pituitary insufficiency.

Standards of Interpretation

Almost all of the glucose tolerance tests are done to establish or eliminate the diagnosis of diabetes mellitus. The value of the postprandial blood glucose for this purpose has been firmly established. Sindoni originally demonstrated that blood drawn one to two hours after a meal is rarely, if ever, elevated in normal persons, in contradistinction to the high values found in diabetics. The serum glucose level is always lower than 120 mg. per 100 m. in normals and always higher than 150 mg. per 100 ml. in diabetics. Here is a procedure which is accomplished without fasting, requires only one blood sample, costs less and is completed in a relatively short time.

In this test as in the above tolerance tests, the patient should be placed on a high carbohydrate diet for at least three days prior to the test so that it may be carried out under optimum standard conditions. While the tolerance tests are done after twelve hours fasting, in the postprandial blood sugar test the patient eats a normal, hearty meal (breakfast, lunch or dinner) and the blood is drawn one to two hours afterwards. Excessive physical activity should be avoided during this period as activity accelerates the rate at which glucose enters muscle cells.

A postprandial serum glucose level of less than 140 mg. per 100 ml. is considered normal. One that exceeds 140 mg. per 100 ml. is abnormal. This test will pick up mild diabetics, i.e., with a moderate persistent elevation of the blood glucose concentration but no glycosuria. Since treatment of these patients will not include insulin but will be directed along lines of weight reduction and other general measures it is immaterial whether diabetes is diagnosed by means of the postprandial blood sugar or by the glucose tolerance test.

Postprandial Blood Sugar Should be Routine

Since many patients with diabetes may exhibit only minor symptoms and may not excrete sugar in the urine, it is wise

to screen all patients by performing a postprandial blood sugar as part of their work-up. It is also of value in following the course of mild diabetics at regular intervals.

The finding of reducing substances in the urine (mellituria) should be followed by a postprandial blood sugar. The finding of a normal postprandial blood sugar eliminates the diabetic syndrome as the cause. Then the possibility of the presence of renal glycosuria, fructosuria, pentosuria or lactosuria should be investigated.

Hereditary Factors

It is universally agreed that there is a large hereditary component in the causation of diabetes mellitus. Recent studies of Stinberg and Wilder suggest that diabetes mellitus is transmitted by a single autosomal recessive gene, d. Individuals who are genetically liable to develop the disease are dd, and those who are not genetically liable to develop it are Dd or DD. It is estimated that 5 per cent of the population of the United States belong to dd. However, because of the variable age of onset and the difficulty of making the diagnosis clinically, only about 1 per cent of the population is recognized as being diabetic. The probability of genetic susceptibility in any family when Dd mates with Dd is the expected 25% of offsprings will be dd. Dd mated with dd gives 50 per cen of offsprings dd and dd mated with dd gives 100% of the offsprings dd. On this assumption it is possible to calculate the probability of any one individual in a family being dd if one knows that certain other relatives in the same family have diabetes mellitus or may be dd .

Cortisone Glucose Tolerance Test

We are still left with the problem of determining which asymptomatic individuals are dd and when they are likely to show symptoms of diabetes mellitus. Fajans and Conn have made progress in this by devising a combined cortisone-glucose tolerance test. They found 19 per cent of 152 relatives of known diabetics showed an abnormality of the usual glucose tolerance test. However if 50 mg. of cortisone was administered eight hours before the test the incidence was 24 per cent. In contrast only 1 of 50 control subjects with no diabetes mellitus in the family demonstrated an abnormality with the usual glucose tolerance test and 1 of 37 control subjects demonstrated blood sugar changes by the cortisone-glucose tolerance test.

Woodyatt and Spetz have suggested that the onset of diabetes mellitus becomes earlier with each affected generation. Thus, if grandfather developed diabetes mellitus at the age of 50, the father may develop symptoms at 30 and the offspring at 10. This is not a consistent relationship between parent and child so the term anticipated should not be used.

Summary

In summary diabetes mellitus is probably transmitted by a simple autosomal recessive gene. Individuals who are homozygous for this gene dd are liable to develop diabetes mellitus but the age of onset is variable. The specific diagnosis is made by the clinical history and the laboratory determination of mellaturia and decreased tolerance to glucose. The best single test for this is the postprandial blood sugar determination.

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Management of Unstable Diabetes*

• Difficulty in the management of diabetes may be due to the patient's outlook on the disease, as well as to faulty management or causes inherent in the disease.

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THE physician's purpose in treatment of diabetes is to minimize its influence on the patient's life. In most cases modern methods of control are entirely adequate to permit diabetics to live lives scarcely distinguishable from those of non-diabetics. In the minority are patients whose diabetes behaves so erratically that it makes anything approaching normal living impossible. This minority is said to have "labile," "brittle," "unstable," or "unmanageable" diabetes. Insulin reactions are often their great daily fear.

When diabetes appears to be unmanageable first thought should be given to finding the reason. Frequently, in my experience, no satisfactory explanation will be found, and patient and physician will have to embark on an often frustrating course of manipulation of diet, hypoglycemic agents, and exercise. Sometimes, however, the physician will be lucky enough to establish the cause and, luckier still, to remove it.

Stable Diabetes Masquerading as Unstable Diabetes

Instability of diabetes may be due to nothing more than the patient's ignorance of how to care for his diabetes at home. This becomes immediately apparent in the hospital where standard management may reveal nothing unusual about the diabetes or response to treatment. In such cases, thorough education of the patient by many means now available usually will tame the disease.

Poor self-management of stable diabetes is not always due to ignorance, however. Conditions at home or work may make it extremely difficult for the patient to follow a regular daily routine. This is true, for example, of patients whose energy output in physical work must vary widely and of those on rotating workshifts. Sometimes a conference with or a letter to the employer will be helpful.

There may also be psychological barriers to achievement of satisfactory diabetic control. These vary in magnitude from poor self discipline and an easily understandable rebelliousness and partial denial of existence of diabetes to use of of the disease for suicide. In such cases it is really not the diabetes but the patient who is unstable, but this may not be immediately evident to the physician. Peck and Peck [1] and Stearns [2] described 5 cases of patients, all adolescents or young adults who attempted to use their diabetes for self-destruction. One succeeded. Another, during a period of seven years was in coma at least forty-two times; adjustment of her environment and superficial psychotherapy stopped this behavior.

Instability of Diabetes Due to Excessive Doses of Insulin

For years it has been known that insulin reactions make diabetes behave erratically for several hours or days. This appears to be due primarily to the stimulating effect of hypoglycemia on release of corticoids and possibly other insulin antagonists. Somogyi [3] appeared to believe that this phenomenon accounts for most cases of diabetic instability. He went so far as to say that every one of his patients who for years had received 30 to

* Presented at the Eighty-first Annual Meeting of the Louisiana State Medical Society, May 9, 1961, in New Orleans.

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150 units of insulin daily was restored to "a status of mild diabetes that can be managed with small doses of insulin or, not infrequently, without insulin".[3] An illustrative case (Figure 1) is that of a

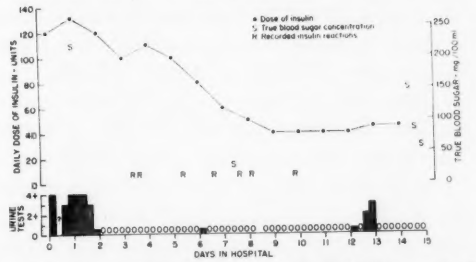
• Dose of insulin
S True blood sugar concentration
R Recorded insulin reactions
DAILY DOSE OF INSULIN - UNITS
TRUE BLOOD SUGAR - mg %
URINE TESTS
DAYS IN HOSPITAL

Figure 1.—Improvement of diabetic control in a 22 year old woman during hospitalization and reduction in insulin dosage.

woman, 22 years of age, who had had diabetes for ten years. For three years before admission to Ochsner Foundation Hospital the diabetes had been poorly controlled. She ate erratically. In spite of injection of 60 units of protamine zinc insulin twice daily results of urinalyses, which she performed three times a day, were rarely negative for sugar and frequently positive for acetone. She had become emaciated, and diabetic neuropathy and nephropathy had developed. In the hospital the dosage of insulin was rapidly reduced and one daily dose of 44 units of NPH insulin satisfactorily controlled the disease. After discharge from the hospital, the daily dosage was reduced another 4 units.

In this case improvement in diabetic control by reduction in insulin dosage, combined with dietary regulation was exceptionally easy to accomplish. More commonly, Somogyi emphasized, great ingenuity in manipulation of diet and insulin is necessary to break the disastrous cycle responsible for "exacerbation of diabetes by excess insulin action".[3]

Instability of Diabetes Due to Concurrent Disease

Reduction in insulin requirement, often of substantial and progressive degree, combined with increasing liability to insulin reactions should make one think of Addison's disease or anterior pituitary insufficiency. Increasing severity of diabetes often accompanies chronic infections or thyrotoxicosis. Diabetes due to pheochromocytoma is notably labile.

An unusual case of diabetic instability associated with concurrent disease is exemplified by a man, 50 years of age, admitted to Ochsner Foundation Hospital on March 5, 1961, with a history of diabetes for the past ten years. Since 1955, he had had symptoms of diabetic neuropathy, chief of these being impotence and orthostatic hypotension. Until March 1960, the diabetes had been easily controlled by a qualitative diabetic diet and injection of 20 units of protamine zinc insulin daily. Then, the diabetes started behaving erratically. He began to have cutaneous infections. Three days before admission epigastric pain developed. Radiologic examination of the upper gastrointestinal tract showed severe gastric retention and at least two peptic ulcers, one of which appeared to penetrate the pancreas. The radiologic diagnoses were confirmed at operation.

One interpretation of this sequence of events is that the instability of the diabetes, beginning one year before admission, was due to intermittent pyloric obstruction, and that the gastrointestinal lesions were asymptomatic, or nearly so, because of coexistence of the diabetic neuropathy affecting principally the autonomic nervous system. Admittedly, the cutaneous infections, as well as the secondary pancreatitis, may have been contributing factors.

Insulin Resistance

Insulin resistance is said to be present when the daily insulin requirement is persistently 200 units or more. The condition as so defined is rare. I can remember having seen only one case. In 1948, Smelo [4] collected only 54 cases from the English medical literature. Insulin resistance has many causes and usually is self-limited in time. Its treatment is not completely satisfactory. According to Berson and Yalow,[5] "approximately half of the pa-

tients will show a significant reduction in insulin requirements following treatment with adrenal cortical steroids." Treatment of some patients with adrenocortical steroids, however, increases insulin requirements.

Instability of Diabetes Due to Unknown Causes

Patients in whom the cause of the disease's instability cannot be found have usually had diabetes since childhood or adolescence, and the disease may have been easily controlled for some years before becoming unstable. Unfortunately, there is no magic formula which will insure satisfactory therapeutic results. What is needed is unrelenting application of basic principles of diabetic management, as well as, at times, a few tricks.

A. **Education of the Patient.** To the limit of his ability, the patient should master the whole subject of diabetes. Knowledge of a few simple facts and technics permits the patient with stable diabetes to live at peace with his disease, but this may not be true of a patient with unstable diabetes. The physician should emphasize the importance of proper technic of administration of insulin, as well as establishment of a regular daily routine with respect to time of administration of insulin, food consumption, urinalyses for sugar, and exercise. The patient should be encouraged to live a life of emotional moderation, and he should be warned of the dangers of insulin reactions. He should at all times carry some form of sugar with him and be ready to use it at the first hint of an insulin reaction. Unusual exertion should be preceded by a feeding. In my opinion, he should be taught to look on moderate glycosuria with an unconcern that would not be permissible in a stable diabetic.

B. **Diet.** Obviously, the diet should be carefully measured. Eating mid-morning, mid-afternoon, and bedtime snacks may be invaluable in preventing insulin reactions and stabilizing the disease.

C. **Insulin.** It seems to be impossible to prepare a satisfactory, brief coherent summary of use of insulin in treatment of unstable diabetes. The following observations are offered in place of one.

1. It is wrong to rely on the belief that any insulin preparation "A" has a duration of action "X" and is maximally active as measured by depression of blood sugar concentration at time "Y" after injection. In the first place, the duration of action of any type of insulin depends partly on size of dose. This is exemplified by the case of the patient reported by Vogl and Youngwirth [6] who was still having insulin reactions six days after injection of 2000 units of protamine zinc insulin, which he had taken with suicidal intent. Secondly, patients vary amazingly among themselves in their response to the same dose of the same preparation of insulin. Hallas-Moller [7] observed that injection of the same depo-insulin into 3 fed patients with diabetes of almost the same degree was followed in six hours by a fall in the blood sugar concentration of about 200 mg. per 100 ml. in one, a rise of about the same degree in another, and little change in the blood sugar in the third.

2. A single daily injection of a single kind of insulin will rarely control unstable diabetes. Single injections of a mixture of insulins (for example, Lente and semi-Lente insulin) sometimes will. More frequently, two injections daily are needed; for example, two-thirds of the total daily dose of insulin given before breakfast and one-third before supper or at bedtime.

3. Protamine zinc insulin, alone or in mixtures, probably should not be used in the treatment of unstable diabetes.

4. Changes in dosage or kind of insulin should be made only rarely at daily intervals.

D. **Oral Hypoglycemic Agents.** Several oral hypoglycemic agents have been used to supplement insulin in treatment of unstable diabetes. Of these, phenethylbiguanide (phenformin, DBI®) has proved most useful. However, the frequency with which it produces gastrointestinal symptoms and lassitude limits its usefulness and even if tolerated by the patient is is

not always effective. Krall and associates[8] report that "the activity appears to start several hours after ingestion and in its present form usually disappears in six to eight hours." This observation can be used as a guide to spacing doses of the compound. Krall's patients who were taking both insulin and phenformin received a daily average of 152 mg. of phenformin and 10 units of insulin. In this group, phenformin had reduced insulin requirement by 64 per cent.

Summary

The following program of management of the patient with unstable diabetes has been suggested: (1) hospitalization; (2) institution of a good standard diabetic regimen; (3) careful search for co-existent disease and treatment of any found; (4) if the diabetes remains unstable: (a) intensive, thorough education of the patient in the technic of injection of insulin, in the pathologic physiology of diabetes, the action of insulin, recognition of insulin reactions, and the diabetic diet; (b) painstaking manipulation by the physician of insulin therapy, diet, and occasionally oral hypoglycemic agents or corticoids, and (5) close supervision of the patient after discharge from the hospital. Moderate glycosuria is, in my opinion, preferable to frequent insulin reactions. In spite of all the patient and physician can do, however, the results of treatment may not be ideal in all cases.

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Who Introduced Nitroglycerine

The following clipping from the April number of the Philadelphia Medical News is of interest in the history of this new remedial agent. It is a point of importance that due credit may be given for what appears to be a valuable discovery, and we gladly republish the following claim made by Dr. Allan M'Lane Hamilton, of New York City:

In an article in the current number of the News it is stated that nitroglycerine is a new remedy. I desire to call attention to the fact that the drug was used many years ago, and articles detailing its physiological effects were written as far back as 1855.

I wish now to claim priority in suggesting its use in the treatment of various nervous diseases, including epilepsy and migraine, in appreciable doses. Dr. A. J. Minor and I conducted a series of experiments in 1875, which were subsequently published. (Am. Pathological J. Feb. 1876)

New Orleans M. & S. J. 10:304 (Oct.) 1882.

Determining the Cause of Death

• The steps by which a diagnosis is established are given, and some statistics on coroner's cases in New Orleans. The necessity of an autopsy is shown.

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NICHOLAS J. CHETTA, M. D.
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FOR each human being who dies there must be a legally documented cause of death. Each year there are approximately one hundred and five million deaths in the United States. Of this number about three hundred thousand qualify under local laws as Coroner's or Medical Examiner's cases. The ratio of medico-legal cases to those not requiring investigation varies widely with local problems. New York City has a yearly average of 76,513 deaths, of which 15,699 require investigation for a percentage of 20.5 per cent. New Orleans during the past nine years has shown a yearly average of 8055 deaths of which an average 3,969 are investigated (a percentage of 49.2 per cent). The death average is comparable on a per capita basis.

It is obvious that the total number of Coroner's Cases is augmented by the Charity Hospital facilities in this area. Last year 25.4 per cent of Coroner's Cases in Orleans Parish represented out of Parish cases reported from Charity Hospital. Of the total cases investigated, the number of cases autopsied has increased from 27.2 per cent in 1951, to 52.72 per cent in 1959. In cases not requiring autopsies the causes of death were established on the basis of hospital records or the medical opinions of attending physicians.

The responsibility for determining that the processes of life have ceased belongs to the physician, and when he is in attendance at the death scene it is simple to set the time and manner of death. The cause may or may not be apparent. Establishing the exact time, manner and cause of death is sometimes a more difficult procedure than is credited, especially by lay opinion.

Signs of Death

The earliest reliable sign of death is the cessation of heart and respiratory movements. In extreme shock, heart and lung sounds may be so feeble as to be barely audible so that careful examination must be made to avoid error. The stethoscope and a fairly good ear are rarely wrong in this regard.

Once metabolism stops, heat production no longer continues and body heat begins to be lost. The rate at which this takes place will depend on a variety of factors, which include environmental temperature, body temperature at the time of death, amount of subcutaneous fat, amount of clothing and exposure to the elements. Sunlight will warm the markedly rigid body as well as the putrefied body recovered from waters several days after death has occurred.

When there is no circulation of blood, stagnation takes place and the blood gravitates to the dependent parts of the body. Post mortem lividity is an indication of the position of the body, and is evidenced by a purplish discoloration of those dependent parts. If the body is moved after lividity is complete, the site of discoloration will not change. If the change in position is made after the process has started but is incomplete, coloration will be noted in more than one area. This process begins within one hour after death.

* Presented at the Eightieth Annual Meeting of the Louisiana State Medical Society, May 4, 1960, in Baton Rouge.

Rigor mortis will usually begin in about the second hour after death and is usually complete in about twelve hours. Beginning with stiffness of the jaw, the process continues caudad in the body until it involves the lower extremities. During the following twelve hours the condition disappears with the same sequence with which it developed. Although rigor is often used as a criterion for setting the time of death, it is subject to great variation. Physical activity, convulsive seizures, febrile states, atmospheric heat and emaciation will accelerate the onset and process of rigor. Cold and heavy body musculature will slow the development as well as the dissolution of rigor. Heat will accelerate the disappearance of rigor. In a cold atmosphere rigidity will persist for days. The bulk of muscle as influenced by the speed of chemical reaction after death determines the rate and progression of rigor.

Putrefaction of tissues occurs once biophysiologico-chemical activity stops. These disintegrative changes are aided by the destructive invasion of bacteria. Depending again upon temperature and atmospheric factors there follows usually within thirty-six hours gas formation, discoloration of tissues, staining of superficial veins and separation of the epidermis. The process continues so that within forty-eight hours there is obliteration of features and gaseous distention of tissues. This may proceed to rupture of soft tissue areas. Mummification seen in hot dry climates is almost never seen in this locality.

The examination of decomposed bodies is not as fruitless as it may seem. Trauma can almost always be ruled out. Poisoning with heavy metals can be determined from scant ash remains and bone surveys. Fractures are easily diagnosed and calcific cardiovascular lesions are frequently identifiable.

Manner of Death

The manner of death is determined on the basis of a careful history as supplemented by information obtained from relatives or other witnesses in attendance at the time of death, and augmented by the personal knowledge of the physician in attendance. The varied clinical pictures are essentially well known and too numerous for the scope of this discussion. The chest pain and shock-like picture of the cardiac; the coma, hemiplegia and other neurological manifestations of the cerebrovascular accident; the hemorrhage of pulmonary and gastrointestinal lesions are but an extremely small number of the grossly apparent signs which are identifiable with terminal episodes of acute or chronic disease. Careful attention to the details of these terminal episodes plus a careful history will aid tremendously in establishing the cause of death.

The presence of trauma must always be considered even under the most obvious medical evidence accounting for death. While the cause of death is frequently known, there is always room for amplification of the diagnosis or the revelation of an obscured clinical entity. In the presence of any doubt or the least dissatisfaction regarding the cause of death it becomes imperative that a complete autopsy be performed. Circumvention of this obligation will only permit the development of doubts in the mind of relatives. It has been the experience of our office that even the most bitterly opposed family and friends of the deceased will yield to the logic of the exact diagnostic information afforded by the autopsy rather than the appeal to their sense of obligation to contribute something to science. This attitude is reflected in the fact that during recent years it is not too uncommon for relatives to initiate the request that an autopsy be performed in the presence of the sudden or unexplained death.

The following tables illustrate the number of cases investigated during the past electoral year for the Orleans Parish Coroner's Office. The distribution is based on the manner of death. The fact that the medical cases do not exactly reflect standard statistics is due to the selective nature of cases which qualify as Coroner's Cases.

TABLE 1
MANNER OF DEATH

Manner		Total	Per Cent of Total
Natural Causes	3,288		
Abortions	2	3,290	76.55
Unclassified		6	.14
Motor Vehicle Accidents		127	2.95
Other Transport Accidents		10	.23
Other Fatal Accidents		272	6.33
Suicides		51	1.19
Homicides		80	1.86
Stillbirths		449	10.45
Under Investigation		13	.30
TOTALS		4,298	100.00

Case Reports

The fact that things are not always what they seem is reflected by the following cases taken at random from the files of the Coroner's Office of the Parish of Orleans.

Case No. 1—F.S., WM 70

This retired old gentleman had been a known diabetic for about fourteen years. He was under treatment for his diabetes and heart disease for many years, by his personal physician. One day he visited his doctor complaining that he noticed some weakness of his right arm and leg since about four days previously. Three hours later he fell in his room at a local apartment hotel and lost consciousness. He was hospitalized and x-rays revealed that he had suffered a linear fracture of the parietal portion of the skull. Neurosurgical consultation was obtained and the consensus of opinion was that the patient had suffered a cerebrovascular accident, and the fall was secondary to his stroke. On the second hospital day he developed Cheyne-Stokes respirations and right-sided convulsions. He ran a febrile course, developed rales in both bases and expired on the ninth hospital day.

An autopsy was performed at the city morgue and the findings revealed a contusion about the right ear. On reflection of the scalp there was considerable hemorrhage under the scalp in the right parieto-occipital region. There was a fracture of the calvarium on the right side extending into the vertex from a comminuted fracture in the temporo-parietal region on the right. When the calvarium was removed there was no extradural blood, but there was 100 cc. of subdural blood on the left side. The brain was compressed and there was flattening of the gyri and obliteration of the sulci. The left temporal lobe was hemorrhagic and soft, and showed hemorrhage within its substance. On cut section, a focus of contusion was seen on the orbital surface of the right frontal lobe. The entire temporal lobe was destroyed by hemorrhage and contusion. There were small petechial hemorrhages in the pons.

Positive findings in the abdominal cavity revealed that the upper pole of the left kidney was replaced by a lobulated mass of tumor tissue which on cut section showed extension into the renal veins.

The lungs showed several small areas of consolidation, which were compatible with pneumonitis. There was one round, circular tumor mass, which represented metastasis from the renal tumor.

Autopsy proved that this man died of this accidental fall, which produced the skull fracture and subdural hematoma and brain injury. The finding of the renal neoplasm was an interesting anatomical finding.

Case No. 2.—E.J., CM 46

While drinking white port wine at a local tavern this negro male was observed to suddenly fall off a bar stool striking his head on the floor. Upon arrival of an ambulance he was pronounced dead and conveyed to the city morgue for an autopsy. There was only minor evidence of a contusion to the forehead. There was no

TABLE 2
AUTOPSIES PERFORMED AND MANNER OF DEATH

		Total Cases	Cases Not Autopsied	Autopsies Performed: Coroner's Office	Autopsies Performed: Hospitals	Autopsies Performed: Total	Per Cent of Cases Autopsied
Natural Causes	3,288						
Abortions	2	3,290	1,812	371	1,107	1,478	44.92
Unclassified		6		5	1	6	100.00
Motor Vehicle Accidents		127	1	124	2	126	99.21
Other Transport Accidents		10		9	1	10	100.00
Other Fatal Accidents		272	85	111	76	187	68.75
Suicides		51	1	50		50	98.06
Homicides		80		80		80	100.00
Stillbirths		449	133	4	312	316	70.38
Under Investigation		13		12	1	13	100.00
TOTALS		4,298	2,032	766	1,500	2,266	52.72

previous medical history. The receipt of an anonymous telephone call relating that the man was struck and knocked off the bar stool by a drinking companion raised further suspicion that death might be traumatic and related to head injury. Police investigation failed to confirm the story of a fight.

An autopsy was performed and the cause of death was established as being due to severe arteriosclerotic heart disease. The left coronary artery showed luminal narrowing of from 40 to 85 per cent. The right coronary artery demonstrated up to 90 per cent narrowing of the lumen in its posterior portion. The myocardium was markedly hypertrophied. The lungs revealed evidence of severe tracheobronchitis. There was no evidence of cranial or intracranial trauma. Death was determined to be due to natural causes.

Case No. 3—M.O., WF 47

This lady has been treated at a local hospital clinic for the past eight years for severe bronchial asthma. On the day of death she developed difficulty in breathing and employed her usual bronchodilator drugs without seeking medical care at the clinic. Later that afternoon she suddenly became more markedly dyspneic and died.

An autopsy was thought advisable because of the age of the patient and her lack of recent medical care. Post mortem examination revealed evidence of bilateral bronchopneumonia and the presence of moderately large pulmonary embolus.

Case No. 4.—S.O., WF 11 Months

This child was dead on arrival at a local hospital. There was a history of the child having been dropped by an older child about two months previously and having suffered a fracture of the right leg. The cast had been removed about two weeks prior to death. A few hours before being brought to the hospital the child reportedly fell from her crib and shortly afterward developed convulsive seizures.

Autopsy revealed a moderate degree of edema and subscapular hemorrhage. Upon removing the calvarium about 40 cc. of clotted and liquid blood was found in the subdural space covering the right cerebral hemisphere. There were no definite lesions of the brain and there was no demonstrable fracture.

Further investigation revealed evidence of cruel and abusive physical treatment by a common-law husband and the case was determined to be homicide.

Intellectual honesty has already been given as a reason for the need for autopsy in medical or so called "natural causes" deaths. The area of suspicion in homicides and suicides is frequently quite obvious. On rare occasions criminal death may prove elusive because of lack of sufficient inspection of the body or attention to details of the manner of death which do not add up to a completely satisfactory answer. Criteria used by our office which arouse suspicion are varied. Autopsies are performed routinely on all cases suspected of homicide, suicide, abortion, traffic fatalities, and industrial accidents. All cases of sudden and unexplained deaths including infants are examined routinely. Other criteria used in determining the need for autopsy include the age of the deceased, unsatisfactory clinical evidence even in the face of a medical history, the interest or suspicion of the attending physician or the request of the immediate family. The locale of death is sometimes a factor since it is well known that some neighborhoods have a higher incidence of cases requiring investigation.

An important consideration in modern times for the need of the medico-legal autopsy is the increasing number of claims arising from deaths involving personal liability and workman's compensation. Nothing will more effectively settle these claims to the best interest of the claimant and the insurance carrier than a documented autopsy protocol. The factor of double indemnity in so many life insurance policies also demands autopsy in many cases. Death in the elderly driver who dies in an automobile accident can frequently pose a problem in deciding whether the exodus was due to pre-existing cardiovascular or cerebrovascular disease, or whether it was due to trauma.

Summary

Criteria for determining the time and manner of death have been reviewed. Experience in the Coroner's Office for the Parish of Orleans shows that in a significant number of cases the presumptive diagnosis is not confirmed by the autopsy findings. Medico-legal demands of litigation procedures as well as intellectual curiosity make the need for the performance of more autopsies mandatory.

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Lemons, the Elixir of Life

Dr. William Schmöle, Professor of Pathology in Bonn, (Medical Record) writes a treatise on "A Scientific Method of Prolonging and Making Comfortable Human Life." He claims that this long and eagerly sought for end may be secured by eating lemons. He bases his method upon the hypothesis that the condition known as old age is produced by a misproportion between the organic framework and inorganic constituents of the human body. In order to prevent this condition and preserve the elasticity of youth, it is only necessary to introduce some substance which will dissolve the excess of mineral matter and allow of its absorption and excretion. Such substances are the inorganic acids, and chief among these citric acid, the next in value being lactic acid. Citric acid is best taken in the form of lemon juice, and it is recommended that a person swallow the juice of from two to eight lemons daily, year in and year out! Such a practice also secures one against the attacks of rheumatism, which afflict old age. It also prevents the degeneration of the arteries and the calcification of the vascular system.

New Orleans M. & S. J. 10:307 (Oct.) 1882.

Emergency and Near Emergency Surgery in Infants With Congenital Heart Disease*

• The many lesions of congenital heart disease are described together with the life-saving measures offered by modern surgery.

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IN the following discussion of surgery for congenital heart disease, in emergency and near emergency situations, I will attempt as a pediatric cardiologist not to usurp the surgeons' mantle as to how such surgery is carried out. Instead I would like to indicate what conditions are amenable to surgical correction or palliation, and how one goes about the management of such cases from the onset of the emergency until the patient is submitted to the surgeon's knife.

I view the care of sick children with heart disease as team work by everyone concerned, from the doctor, who first sees the patient through the pediatric cardiologist, to the surgeon, who, we hope, finally does something to favorably influence an otherwise dismal picture. Like any team, this team also will function better if all members are equally familiar with the plays. It is not my purpose to make pediatric cardiologists of you, and accordingly, little will be said of the specific technical procedures used in the catheterization laboratory except to mention them by name.

Emphasis will be placed, (1) on how you can suspect that some congenital heart lesion is causing the patient's difficulty, (2) more or less identify the type of the defect, (3) treat the immediate problem, (4) offer a general prognosis, especially regarding the possibility of surgery, (5) seek competent assistance for further management, from the cardiologist for diagnosis and interim care and from the surgeon for definitive treatment.

TABLE 1
APPROACH TO EMERGENCIES IN CONGENITAL HEART DISEASE

1. Suspect heart disease
2. Identify the type of defect
3. Treat the immediate problem
4. Offer a general prognosis
   a. Possibility of surgery?
5. Seek competent assistance for further management
   a. Diagnosis: **Pediatric cardiologist**
   b. Interim care: **Pediatric cardiologist**
   c. Definitive: **Cardiac surgeon**

TABLE 2
EMERGENCIES AND NEAR EMERGENCIES ASSOCIATED WITH CONGENITAL HEART DISEASE

- Congestive heart failure
- Hypoxemia
- Respiratory obstruction

In general there are three classes of emergency or semi-emergency situations associated with congenital heart disease. As shown in the table, these are congestive heart failure, hypoxemia and respiratory obstruction.

TABLE 3
CONGESTIVE HEART FAILURE

I	Myocardial anoxia
II	Obstruction to blood flow
III	Left to right shunt
IV	High output failure

The greatest problem from the point of view of numbers is that of congestive heart failure. The patient may or may not respond to medical mangement. The response will determine further management. Any episode of congestive heart failure even with excellent response to

* Presented at the Twelfth Annual Meeting and Scientific Session of the Louisiana Heart Association, Confederate Memorial Hospital, April 26, 1961, Shreveport, La.

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digitalis alone should be considered at least a near emergency and a signal that the time has come for definitive diagnosis.

Our practice is to carry out specific cardiac diagnostic studies as soon after the patient's condition stabilizes as possible, the degree of urgency depending of course on the level of response to medical therapy. In general, the younger the child at the onset of failure, the sooner we would prefer to go ahead with cardiac catheterization and/or other indicated procedures. I feel that young infants should certainly be studied within one month of the initial episode. Heart failure at such tender age invariably indicates a severe lesion.

Not infrequently, after apparent good initial response these little patients will become increasingly more difficult to manage. They may go bad at varying rates, sometimes so rapidly that diagnostic studies are impossible. One may look on the intervening time as a period of grace. After diagnosis has been established and the feasibility of surgical correction or palliation considered, the patient is managed medically until the optimum time for such surgery. Should the patient's condition deteriorate so that true emergency surgery must be considered, it can be rationally approached, if a complete and proper diagnosis is in hand.

TABLE 4
CONGESTIVE HEART FAILURE

I Myocardial anoxia
- Anomalous left coronary artery
- Aortic stenosis

In anomalous origin of the left coronary artery, the left anterolateral wall of the heart is supplied by blood which is flowing into the pulmonary artery at a fairly rapid rate. Apparently the main cause of difficulty here is the low pressure in the vessel due to its connection with the pulmonary artery, and this rapid drain off results in improper oxygenation of the myocardial musculature along its course. Aortic stenosis is mentioned here for completeness since an anoxic problem does exist and is largely responsible for such things as the sudden death for which this lesion is notorious. It will be discussed more fully later.

TABLE 5
ANOMALOUS LEFT CORONARY ARTERY

Suspect:
- Painful episodes (especially in C.H.F.)
- Shock-like episodes (especially in C.H.F.)
- No other good explanation

Dx helpers:
- EKG—infarction pattern L.V.H. pattern
- X-ray—Left ventricular enlargement Generalized E.H.

Special studies:
- Often unnecessary
- Retrograde aortogram

Surgery:
Ligation of left coronary at its origin from m.p.a.

As indicated in the table the combination of painful episodes with a shock-like picture of clammy skin, pallor, or cyanosis following periods of increased activity, even such minor activities as feeding, is very suggestive. When combined with congestive heart failure, it is almost diagnostic.

Electrocardiogram and x-ray are confirmatory, the EKG showing a pattern of anterolateral myocardial ischemia (deep Q waves, elevated ST segments and inverted T waves) that should not be misinterpreted as left ventricular hypertrophy.

The x-ray shows a dilated left ventricle. If too much damage to myocardial muscle has not already occurred, ligation of the coronary artery at its point of origin from the pulmonary artery, may allow restoration to a fairly normal state. Several patients are now alive following this procedure but a long term follow-up is lacking.

TABLE 6
CONGESTIVE HEART FAILURE

II Obstruction to blood flow
A—Right sided
- Pulmonary artery coarctation
- Pulmonary stenosis
- Tricuspid stenosis or atresia

The right-sided obstructive lesions are typically associated with radiologically clear lung fields.

TABLE 7
ISOLATED PULMONARY STENOSIS

Suspect:
- Congestive heart failure
- Typical "diamond shaped" murmur

Dx helpers:
- EKG:
  —marked R.V.H.
  —R.A.H. ("P pulmonale")

Special studies:
- Right heart catheterization (if time permits)
- Cineangiocardiography (if time permits)

Surgery:
- Pulmonary valvulotomy
  —Brock
  —Open heart with pump oxygenator
  —Inflow occlusion and hypothermia

Isolated pulmonary stenosis is by far the most frequent of this group being most often valvular; but not infrequently infundibular stenosis also occurs, either in combination or alone. Congestive heart failure associated with the typical diamond shaped murmur and with electrocardiographic evidence of marked right ventricular hypertrophy is extremely suggestive. When the X-ray demonstrates radiologically clear lung fields, and/or poststenotic pulmonary arterial dilatation, one may feel fairly certain.

As indicated in the table atrial hypertrophy is present, frequently with giant P waves which have come to be named "P pulmonale" because of this association. If the patient is not in extremis and time permits, right heart catheterization with cineangiocardiography is carried out. Surgery available for this lesion consists of opening the obstruction. In emergency situations in small infants, it is my feeling and has been the experience of the L. S. U. group that Brock's procedure is the safest and is usually satisfactory. Other procedures have been used and are probably preferable in older children and in patients not in extremis.

TABLE 8
TRICUSPID STENOSIS OR ATRESIA

Suspect:
- Congestive heart failure
- Little or no murmur
- Deep cyanosis

Dx helpers:
- EKG: Left axis deviation
- Left ventricular predominance
- X-ray: Bronchovascular markings decreased
  —cut-away right ventricle in L.O.A. projection

Special studies:
- Right heart catheterization
- Selective cineangiocardiography
  Emergency: Peripheral angiogram

Surgery:
- Potts or Blalock
- Atrial septectomy
- Right heart by pass

Triscupid stenosis or atresia may or may not result in congestive heart failure when the patient is first seen. The combination of deep cyanosis, little or no murmur, electrocardiographic evidence of left axis deviation and left ventricular predominance with x-rays showing decreased vascular markings is practically diagnostic. On the left anterior oblique projection, x-ray wise, the right ventricle may appear to be cut away, evidence that it is hypoplastic. Right heart catheterization with selective cineangiocardiography will allow complete diagnosis.

In very sick patients or small extremely cyanotic infants where one is unwilling to do a complete procedure, peripheral cineangiocardiography may give enough information to allow surgical palliation. There is no conceivable way to correct this lesion, but if a large enough pulmonary artery exists, a shunt operation may be done, connecting the systemic and pulmonary circulations. Should congestive heart failure be present and the angiogram indicate a too small atrial septal opening, then combining a Blalock with a modified Blalock-Hanlon atrial septectomy may succeed where a shunt alone would not.

The left sided causes of obstructive failure are listed in Table 9, coarctation of the aorta, aortic stenosis, mitral stenosis, cor triatriatum, obstructive anomalous pulmonary venous return (which is ordinarily

TABLE 9
CONGESTIVE HEART FAILURE

II Obstruction to blood flow
B. Left sided
- Coarctation of aorta
- Aortic stenosis
- Mitral stenosis
- Cor triatriatum
- Pulmonary venous stenosis
- Obstructive anomalous pulmonary venous return
  —Transhepatic ("subdiaphragmatic")
  —Others

transhepatic, but obstruction may occur at other places).

Coarctation of the aorta is a situation in which the aorta is narrowed to such a degree as to cause obstruction. One ordinarily finds upper extremity hypertension

TABLE 10
COARCTATION OF THE AORTA

Suspect:
- Upper extremity hypertension
- Weak to absent femoral pulses
- Lower extremity hypotension
- Typical murmurs

Dx helpers:
EKG:
- Left ventricular hypertrophy
- Right ventricular hypertrophy in young infants

X-ray:
- Left ventricular enlargement
- "E" sign
- Notched ribs

Special studies:
- Double arterials
- Retrograde aortography
  Arterial catheterization

Surgery:
- Excision and reanastomosis

combined with weak to absent femoral pulses, and lower extremity hypotension. A more or less typical pansystolic murmur is heard best posteriorly along the left spinal border.

Spencer and others have shown that with lumen sizes of 2.5 mm. or smaller continuous murmurs can be heard. The electrocardiogram demonstrates left ventricular hypertrophy; however, in very small infants the electrocardiographic pattern may be more that of right ventricular hypertrophy. The x-ray demonstrates left ventricular enlargement and in older children the typical "E" configuration of the barium esophogram and notched ribs may be noted.

Special diagnostic studies such as double arterials, retrograde aortography, and arterial catheterization may be carried out. Surgery consists of excision and reanastomosis.

TABLE 11
AORTIC STENOSIS

Suspect:
- Typical murmur
- Typical thrill
- History of acyanotic syncopal attacks

Dx helpers
X-ray:
- Poststenotic dilatation
- Left ventricle enlarged

EKG:
Left ventricular hypertrophy
Left ventricular "strain"

Special studies:
- Left heart catheterization
- Cineangiocardiography
- Trans-septal catheterization

Surgery:
Valvulotomy

Obstruction at the aortic valve may present as acute congestive heart failure. The presence of congestive heart failure in a patient with a high right parasternal systolic murmur and a thrill that transmits into the neck presents very little problem in diagnosis; however, in small infants, the murmur is usually along the left parasternal border and this does present a problem. The combination of a systolic murmur that could be aortic with the history of acyanotic syncopal attacks is most suggestive.

The x-ray shows enlargement primarily of the left ventricle and may show some poststenotic dilatation of the aorta. EKG ordinarily shows left ventricular hypertrophy and moderate to severe degrees of strain as indicated by ST segment and T wave changes. Unfortunately the electrocardiogram is not infallible in this respect and a small number of patients have been described with severe aortic stenosis causing myocardial ischemia, whose electrocardiogram had not shown progressive increase in strain pattern. Because of this, any patient with possible aortic stenosis

should have a definite diagnosis established and be closely followed if aortic stenosis is the diagnosis.

This lesion can be definitely identified by several methods such as left heart catheterization with selective cineangiocardiography. The left heart may be entered ordinarily in a retrograde fashion through the arterial system from above or it may be entered from the right atrium by means of transeptal catheterization.

Surgery consists of valvulotomy and is ordinarily quite gratifying. A certain number of these patients however, especially those in failure in infancy suffer also from endocardial fibroelastosis and in these cases the outcome is not often favorable.

TABLE 12
PULMONARY VENOUS OBSTRUCTION
(Mitral Stenosis, cor triatriatum P.V. stenosis, obstructed anomalous P.V. return)

Suspect:
- Fairly normal 1st 6 mos.
- Acyanotic "weak spells"
- Heart failure with small heart

Dx helpers:
EKG: Right ventricular hypertrophy
X-ray: small heart
- Feathery pattern of P.V. engorgement

Special studies:
Right heart cath.
Selective cineangiocardiography

Surgery:
Complete Repair
- Heart-lung machine
- Deep hypothermia

The remaining left sided obstructive lesions may be classified under a single heading, roughly titled pulmonary venous obstruction. The syndrome varies considerably but has recently been described rather neatly in its typical form by Dr. Catherine Neill of Johns Hopkins.

These infants may be fairly normal for the first six months except for occasional acyanotic weak spells. Around six months of age, these spells increase in number and the patient may present with clinically typical heart failure. In my own experience this has occurred most often at two to three months of age. X-ray however may reveal a small to only slightly enlarged heart. Electrocardiogram demonstrates right ventricular hypertrophy. In patients with mitral obstruction there will be left atrial enlargement evident both radiologically and electrocardiographically. Cor triatriatum may give similar findings.

The pulmonary vascular pattern on x-ray has a typical feathery appearance of pulmonary venous engorgement. Selective cineangiocardiography carried out during right heart catheterization with the injection of radio opaque dye into the pulmonary artery will demonstrate the point of obstruction.

Insofar as surgery is concerned, complete repair is now possible with the heart lung machine. Deep hypothermia utilizing the heat exchanger may be used also depending upon the surgeon's discretion at the time of operation.

TABLE 13
CONGESTIVE HEART FAILURE

III Left to right shunts

Precardiac
- Anomalous P.V.

Cardiac
- Atrial septal defect
  —Ostium secundum
  —Ostium primum
- Ventricular septal defect
- Combinations of above
- L.V. → R.A. syndrome

Post cardiac
- Patent ductus arteriosus
- Ruptured sinus-valsalva aneurysm
- Aortico—pulmonic septal defect
- Coronary artery fistulae
  —Rt. ventricle
  —Rt. atrium
  —Pulmonary artery

The most frequent cause of congestive heart failure in congenital heart disease is what we term left to right shunts. In this situation because of some defect, blood passes from the higher pressured systemic circuit to the lower pressured right-sided circuit in varying amounts depending on the severity of the lesion.

This results in re-circulation of blood through the right-sided and pulmonary circuit. In its most severe form the syndrome is characterized by repeated pul-

monary infections, gradually increasing congestive heart failure, and eventually death unless some action is taken.

I have divided the abnormalities as shown in the table into precardiac, cardiac and postcardiac. The only true cyanotic patient in this group is the precardiac lesion of total anomalous pulmonary venous return. The others, atrial septal defect, combinations, the left ventricular to right atrial shunt, patent ductus, ruptured sinus of valsalva aneurysm, aortico-pulmonary septal defect, and coronary artery fistula all have a certain similarity. Any of these situations in severe heart failure, however, may appear cyanotic.

TABLE 14
VENTRICULAR SEPTAL DEFECT

Suspect:
- L→R shunt
- Pansystolic murmur, LPSB
  —may be absent in C.H.F.

Dx helpers:
EKG: Left ventricular hypertrophy
? R.V.H., varying degrees
X-ray:
Left ventricular enlargement
Left atrial enlargement—if so rules out atrial septal defect
Increased bronchovascular markings

Special studies:
- Right heart catheterization
- Selective cineangiocardiography

Surgery:
Banding P.A.
Complete repair
- Heart-lung machine

Because of the similarity, we will discuss first the most frequent and typical lesion of this group with a word as to those points suggestive of other lesions. As shown in the table this is a ventricular septal defect. Typically these patients have a pansystolic murmur along the lower left parasternal border which may be in young infants widely transmitted. In severe congestive heart failure there may be little or no murmur.

The x-ray will demonstrate left ventricular enlargement, possibly left atrial enlargement and always moderate to severe degrees of increased bronchovascular markings. Left atrial enlargement mitigates against atrial septal defect.

Electrocardiogram demonstrates left ventricular hypertrophy and varying degrees of right ventricular hypertrophy, depending on age and the degree of pulmonary hypertension. These patients, not infrequently present a history of having had repeated episodes of so called bronchitis or bronchopneumonia. Typical asthmatic wheezes may occasionally be heard. During the early stages, gross congestive heart failure may not be evident. However, as time passes, possibly with each succeeding pulmonary infection, the heart failure becomes more and more evident. When severe congestive heart failure is present, this is indeed an emergency.

After stabilization has been obtained, diagnosis may be established by right heart catheterization with selective cineangeocardiography. It is not usually possible to control these very sick infants with medical therapy alone and some attempt at reducing the load must be made. Some people feel that complete heart repair using the heart machine is feasible, however the widest experience indicates that it is best in these small infants, who usually range from four to eight months in age, to band the pulmonary artery. This procedure consists of placing a tape around the pulmonary artery and constricting it so that the blood flow to the lung is reduced. In effect what is done is to convert the patient into a tetralogy of Fallot. It is usually possible to adjust the size of the orifice so that a right to left shunt with resultant cyanosis does not occur, creating a so-called balanced tetralogy. This procedure has been used with considerable success at L.S.U., where the operation in its present form originated, and in other institutions throughout the country. As these patients become older they invariably become cyanotic and eventually require complete repair.

Atrial septal defects do not ordinarily present as emergencies, and when they do, they are usually of the ostium primum type. Differentiation from ventricular septal defect under these circumstances is ordinarily rather difficult on purely

TABLE 15
ATRIAL SEPTAL DEFECT

Suspect:
- L→R shunt
- Pulmonic murmur

Dx helpers
EKG:
- Primum: R.V.H., L.A.D.
- Secundum: R.V.H., RBBB.

X-ray:
- Increased bronchovascular markings
- P.A. prominence
- Right atrial prominence

* "Atrials" in trouble in infancy & childhood most often are primum type

Special studies:
Right heart catheterization

Surgery:
Complete repair
- Hypothermia
- Heart-lung machine

clinical grounds. The occurrence of a typical electrocardiogram of right ventricular hypertrophy and left axis deviation may be very helpful. The differentiation may be easily established by means of right heart catheterization. These lesions are best completely repaired, but such is rarely done on an emergency basis.

TABLE 16
POST CARDIAC SHUNT
(P.D.A., A.P.S.D., C.A.F., Rupt S.V.)

Suspect:
- L.→R. shunt
- Bounding pulses

* Sudden onset:
- Ruptured sinus of valsalva

Dx helpers:
EKG:
- L.V.H. - Diastolic
- R.V.H. - Less frequent

X-ray:
- Enlargement—L.V. & ? L.A.
- Prominent P.A. segment
- Increased bronchovascular markings

Special studies:
- Retrograde aortography
- Rt. & Lt. heart cath.
- Selective cine.

Surgery:
- Complete repair
- Ligation (C.A.F., P.D.A.)

Post cardiac shunt, presents similarly to the intracardiac shunt but has some particular peculiarities which will usually allow a clinical differentiation. The first of these is bounding pulses; second is the persistence of the murmur into diastole. The association of sudden onset of heart failure in a previously well individual with bounding pulses and a continuous murmur is more or less diagnostic of a ruptured sinus of valsalva. Surgery for these lesions consists of complete repair of the defects and ligation of the abnormal vessels.

TABLE 17
ANOMALOUS PULMONARY VENOUS RETURN
(Complete, with exception of obstructive types)

Suspect:
Congestive heart failure
Cyanosis
L.→R. shunt

Dx helpers:
E.K.G.:
Right ventricular hypertrophy
Right atrial enlargement

X-ray:
R.V. enlargement
R.A. enlargement
Widening of mediastinum ("snow-man")
Increased bronchovascular markings

Special studies
Rt. heart catheterization + selective cineangiocardiography
—P.A. injection

Surgery:
Complete repair
—Heart lung machine

Anomalous pulmonary venous return as stated before should be suspected when a cyanotic left or right shunt remains so even after full digitalization. If there is a pulmonary murmur, right ventricular and atrial enlargement or mediastinal widening one can be quite suspicious. The true "snow man" shadow is unusual in small infants. The lesions can be readily identified in the catheterization laboratory and fully corrected with modern surgical methodology.

The fourth group of causes for congestive heart failure in congenital heart disease, that of high output failure, has only one true member, transposition of the great vessels. In this condition the primary abnormality is that of abnormal conotruncal rotation such that the aorta originates from the right ventricle, whereas the pulmonary artery originates from the left ventricle. Obviously, some associated

TABLE 18
TRANSPOSITION OF GREAT VESSELS

- Suspect:
  - Intense cyanosis in infancy
  - C.H.F.
  - Few murmurs
- Dx helpers:
  - EKG:
    - R. & L.V.H.
    - Deep precardial "S"
  - X-ray:
    - Enlarged heart
    - Narrow waist
    - Widens in L.O.A.
    - Increased bronchovascular markings
- Special studies:
  - Rt. heart cath.
  - Selective cine.
  - Peripheral cine.
- Surgery:
  - Atrial septectomy
  - P.A. banding
  - Complete repair.
    - Deep hypothermia
    - Pump-oxygenator

opening to allow at least a partial mixing must be present in order for the infant to survive.

About three-fourths of these patients will have a ventricular septal defect. About half of them will have either pulmonic stenosis or pulmonary vascular obstruction so that diagnosis and treatment are further complicated. The patients of which I speak here are those with the pure syndrome in whom the only defect, other than transposition of the great vessels is a small atrial defect or only a patent foramen ovale. It is these patients, presenting ordinarily anywhere from birth to three months of age who represent the greatest emergency.

In the transposition group because the two circulations are more or less separate, deoxygenated blood is being recirculated throughout the systemic circulation. The body's normal protective mechanisms result in an expanded blood volume and increased cardiac output. This increase in output affects both sides of the heart and results in the appearance of increased bronchovascular markings, or increased pulmonary blood flow, whereas right and left heart output will not be so greatly different. The necessity to increase the cardiac output results in both right and left sided failure. The presentation of an infant with intense cyanosis, congestive heart failure and few murmurs should lead to an immediate suspicion of transposition.

The electrocardiogram may show left or right ventricular hypertrophy and frequently shows a deep S wave across the precordium.

The x-ray is very suggestive in that it shows an enlarged heart with a very narrow waist which widens in the left anterior oblique position. The bronchovascular markings are increased.

The diagnosis can be readily established and the associated lesions properly identified by right heart catheterization with selective cineangiocardiography. In critical situations a peripheral cineangiocardiogram may be sufficient to allow for emergency surgery.

A rational approach to surgery would seem to be a procedure which would allow for more complete mixing. In very sick infants, I believe the procedure of choice in most institutions should be an atrial septectomy by the modified Blalock-Hanlon method. When a ventricular septal defect is present it may be advisable to band the pulmonary artery. Recently deep hypothermia using the heat exchanger and the pump oxygenator, by lowering the patient's temperature to the vicinity of 15 to 16 degree centigrade, has allowed the removal at that temperature of all of the encumbering apparatus associated with the heart lung machine. This allows the surgeon to work in a dry field and has allowed complete correction of these lesions. By complete correction I mean complete functional correction. The great vessels are not transposed but the venous return is transposed, so that when the procedure is completed the right ventricle functions as the systemic ventricle and the left ventricle functions as the pulmonary ventricle.

The addition of this new technique to the surgeon's armamentarium promises to bring even more so called uncorrectable lesions into the realm of feasible surgery.

Summary

In summary, I would like to say that it is no longer possible to look at a sick child with congenital heart disease, as an unfortunate circumstance about which nothing can be done. It is now mandatory that any sick infant or child in whom the possibility of congenital heart disease exists, be given the benefit of proper diagnosis, so that proper surgical correction or palliation may be undertaken if possible—and it is usually possible.

Vaccination vs. Protective Inoculation

Pasteur having conducted a series of experiments with regard to a certain form of contagious disease in cattle, the medical journals almost universally speak of the operation as charbon-vaccination. It is time that this abuse of the term vaccination were corrected. What Lady Mary Wortley Montague introduced into England as a prophylactic measure against small-pox was inoculation with small-pox. It had its inconveniences; it was uncertain in its effects, spreading the disease, and occasioning deaths now and then; but still it was based on experience, and served its purpose. When vaccination came, however, inoculation ceased by common consent, and it is now generally interdicted by law. Whatever refinements Pasteur and his school introduced with regard to the cultivation of the contagium vivum, they still propagate the disease by using the morbid products of the identical affection. All that they claim is that by inducing a mild attack of the disease which the animal survives, it is shielded from subsequent attacks by the self-protective power of the malady itself. Now, vaccination in the human subject **never** causes small-pox. **Medical Times**, October 21.

New Orleans M. & S. J. 10:442 (Dec.) 1882.

Principles and Problems of Phalangeal Fracture Treatment*

• Return of function to a fractured finger is of great importance for it may affect the means of earning a livelihood in those who have suffered such an injury.

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TO understand the principles involved in the treatment of fractured phalanges, a thorough knowledge of the function of the upper extremity is needed. The phalanges are the terminal devices man utilizes to earn his living, and as one means of expressing himself. Thus, these functions are necessarily instinctive or learned, and are tempered by individual need and concern. Fractures of the phalanges are therefore important and may become serious fracture problems if they are not recognized initially and properly treated. The patient and physician should not adopt a fatalistic attitude, accepting poor end results as inevitable with phalangeal fractures, since good results can be obtained if the fundamental principles involved in treatment are understood and proper treatment is carried out.

Anatomic Structure

What is different about fractures in the phalanges? To begin with, the anatomy is peculiar in several ways. On the volar surface of the phalanx are tendons that are tightly enclosed in a sheath that does not allow impingement on the space without impairing function. Each tendon has separate but synergistic function and a difference in excursion.

An extremely complicated structure is the extensor mechanism. The long extensor enters the region of the proximal phalanx dorsally and is immediately anchored centrally by transverse fibers from the intrinsic muscles. Trailing distally, these fibers are transverse to oblique and give way to distinct lateral bands on either side of the proximal phalanx.

The lateral bands arise from the intrinsic muscles. They are loosely interlaced to the central tendon as far as the insertion at the base of the middle phalanx. The lateral bands at this joint are also anchored on the volar surface by rather strong fibers, which are attached to the flexor sheath. Distal to the proximal interphalangeal joint the flat bands gently converge dorsally to form a single, flat, tendinous structure that inserts at the base of the distal phalanx. It becomes apparent from a description of this structure that fractures of the phalanges require meticulous immediate treatment and after care.

Another facet of the anatomic structure of the finger to be considered is the insertion of tendons on the phalanges. The proximal phalanx has the interosseous insertion which flexes the proximal phalanx. The middle phalanx has the sublimis insertion proximally to give flexion to this phalanx. Distally, the profundis inserts at the proximal aspect of the distal phalanx to give distal phalangeal flexion. These insertions affect position of fracture fragments, as well as the extensor insertions at the proximal part of the middle phalanx dorsally and the proximal part of the distal phalanx dorsally, in combination with the effects of gravity.

Functional Position

What is the functional position and why so much concern? The functional position is the position held with the wrist at 25°

* Presented at the Eighty-first Annual Meeting of the Louisiana State Medical Society May 8-11, 1961 in New Orleans.

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dorsiflexion and the metacarpophalangeals and proximal interphalangeals at 45° flexion. The metacarpophalangeal joint and collateral ligaments are taut when the former is flexed to 45°. The middle interphalangeal joint and collateral ligaments are to a lesser degree taut when flexed, but this is not the only important structure causing proximal interphalangeal joint tightness.

The second structure at the proximal interphalangeal joint is composed of the lateral bands that normally sublux volarward, pulled so by Kaplan's fibers when the joint is flexed. If the lateral bands are not allowed to sublux volarward, they coalesce and become an inherent part of the central slip. These important structures, the collateral ligaments and the lateral bands which must sublux with the proximal interphalangeal joint in flexion, are the major factors in permitting finger motion in flexion, as long as the extensor mechanism is not damaged. If the central slip of the extensor is damaged and sticks down proximal to the joint involved, this obviously precludes flexion of the joint distal to this. The biological rule which dictates that all movable soft tissues, if left relaxed, will tighten up, is the simple essence of the functional position. Also the finger flexors and extensor are at the proper fiber length to start flexion and extension most advantageously.

Methods of Fixation

The most serious mistake that can be made in managing a fractured phalanx is to try to employ one method of fixation for all fractures. If one considers the anatomy and the various types of fractures at each level, the best solution usually becomes apparent.

Stable fractures, that is, stable in an axial direction (Fig. 1), can usually be managed by splinting to include a normal finger that helps to control alignment and and rotation. Usually, immobilization of the wrist in the functional position helps to maintain the desirable position of the

Figure 1.—(a) Epiphyseal fracture dislocation. (b) Reduced and stable. Held with splint and tape.

phalanges, and also the splint remains much more stable.

Oblique fractures are best managed by open reduction and fixation with multiple

Figure 2.—(a) Compound fractures of phalanx with multiple extensor lacerations. Quite unstable. (b) Fracture reduced and transfixed with multiple Kirschner wires extensor tendons sutured. (c) Fractures healing six weeks after injury.

small wires, followed by splinting in a functional position (Fig. 2).

An alternate method for oblique fractures is skeletal traction in a functional position (Fig. 3), but constant care is needed to insure adequate and continued reduction until stability occurs. Figure 3b shows poor position of fracture.

The most difficult fracture to treat is the comminuted one that defies internal fixation and must be managed by skeletal traction with the finger splinted in a functional position (Fig. 4a & b). When skeletal traction is utilized, dexterity is needed to align fragments at the various levels, as loss of bony substance and muscle pull are continuous deforming factors. Immobilization of the wrist is believed to be indicated in most finger splints if the fractures are unstable.

Lastly, a word of caution should be sounded about malrotation and lateral deviation as a cause of embarrassing functional impairment, such as the crossing of the fingers when flexed.

Restoration of Function

It is important to know when attempts should be made to begin finger motion. Such criteria as radiologic evidence of healing of the fracture must be discarded as an indication for beginning motion. By early mobilization of inherently stable fractures without loss of substance, we must depend primarily on fibrous tissue healing of the degree reached by about three to four weeks, combined with what fracture healing has ensued. However, this usually is enough stability to start finger motion.

Fractures that are inherently unstable and are comminuted usually are beginning to coalesce at three to four weeks, so that one may at least begin to mobilize adjacent joints. Intermittent splinting must be continued to prevent further deformity and drift of the distal part. The more comminuted the fracture and the less anatomic apposition of the fragments, the more difficult it is to hold the fragments and restore function in the involved area and of the finger distal to the fracture.

Figure 3.—(a) Oblique, unstable fracture. Treatment with traction. (b) Volar angulation of distal fragment; realignment was indicated.

The healing mass is directly related to the fracture surface involved. The healing mass envelops all surrounding structures to give a fibrous fracture mass that is initially inert functionally and must follow well-known healing evolutionary processes. There is first the violent soft tissue reaction of swelling and hemorrhage, which gradually subsides. Coincidentally, the fractured ends are being

united by the process of fracture healing, so that by the end of three or weeks fibrous tissue is interspersed with osteoid and there is some endosteal healing. Gradually, the fibrous tissue mass is reduced in bulk as the fracture ends are solidified by new bone, which eventually remodels as the stresses indicate. The more anatomically reduced a finger fracture is, the better the function of the finger.

Figure 5a illustrates an oblique fracture. Figure 5b shows the healed fracture with incomplete reduction; the patient has 25° loss of flexion at the proximal interphalangeal joint.

The last chance to salvage a fractured finger is about three weeks after fracture. Then begins the golden period for functional recovery. To succeed, there must be active motion of appropriate type and amount, to include functional splinting when active exercises are failing to accomplish the desired results early.

When the metacarpophalangeal joint is being mobilized, the patient must concentrate on this joint, to avoid automatic

Figure 4.—(a) Compound comminuted fracture with extensor tendon lacerations. Treatment with tendon suture and traction. (b) Fractures healing in good position.

flexion of the proximal interphalangeals, thus losing needed power of the long flexor that can be utilized if the proximal interphalangeal joints are held stiff and all the excursion is carried out at the metacarpophalangeal joint.

If help is needed, a Bunnell knuckle bender splint is indicated. Rarely is the metacarpophalangeal joint difficult to extend. If it is, and help is needed, a Bunnell reverse knuckle bender splint will assist. The proximal interphalangeal joint is mobilized best by use of a block of wood to grasp so that it fits in the base of the palm, then stops just short of the midpart of the proximal phalanx.

If additional help is required in flexing the phalangeal interphalangeal joint, a Bunnell double knuckle bender splint is needed so that metacarpophalangeal and

phalangeal interphalangeal flexion may be carried out, gradually and passively, with active motion carried out by use of the block. The combination of both is best. If help with phalangeal interphalangeal joint extension is needed, a knuckle bender splint with extension outriggers will usually give enough passive help so that with passive extension of the splint, active entension of the long extensors and intrinsics will usually result in restoration of the needed motion.

The last phase of hand injury problems occurs in the occasional individual with sympathetic overactivity that causes continued swelling, pain, and increased sensitivity of the skin. All these factors contribute to the coalescing and nonfunctioning of vital structures. Too little has been discussed about this abnormal continuation of a normal phenomenon in trauma. The increased sympathetic activity is part of the physiological response to trauma. If prolonged, it occurs in all degrees, and goes under such guises as Sudek's atrophy, minor causalgia, and sympathetic dystrophy. Prolonged sympathetic overactivity may be controlled by prevention and control of infection, and early mobilization of all upper extremity joints not involved in the injury, as well as the involved digits. Sparine® in doses up to 100 mg., every three to four hours, combined with long-acting codeine or other analgesic preparations, such as Donnagesic #1 or #2®, is also helpful. These drugs are inducted only in the presence of definite sympathetic overactivity, and then they should be used adjunctively with guided physical therapy and proper dynamic splinting. The patient with arthritic joints will most certainly have trouble when mobilization of the joint is begun (Fig. 6). This problem necessitates earlier mobilization.

Figure 5.—(a) Oblique unstable fracture of fourth proximal phalanyx. (b) Healed fracture of fourth proximal phalanyx with lack of anatomic reduction. Treatment with traction.

Conclusion

In conclusion, it should be pointed out that this necessarily brief discussion could not include all possible fractures in each

Figure 6.—Degenerative articular changes of interphalangeal joints.

given case. However, the essentials utilized in the care of phalangeal fractures have been covered. Continued close evaluation of the end results by the surgeon who treats the patient will necessarily keep him concerned about his method of treatment, as final function in fractured fingers almost never approaches that of a normal digit.

Discussion

Dr. Harry Morris (New Orleans): Dr. Brooks is to be congratulated for his precise presentation of the fundamentals of treatment of fractures of the phalanges. Our contemporaries at the University of California inform us that for only two reasons does a human being exceed in efficiency the lower animals. First is the ability to think logicaliy and second is prehension or the ability to oppose the digits in grasping. It is because of the importance of the hand, with its individual digits, to the economic and functional capacity of the individual that we are constantly striving to improve the end results of trauma to the digits by intelligent early treatment based on fundamental anatomic knowledge and principles. Not too long ago treatment of these then considered insignificant phalangeal fractures was relegated to the "lowest man on the totem pole," and the time and strength of the more experienced surgeons were conserved for taking care of the "big stuff." However, we have been shown by a dedicated group of physicians, principally orthopedic surgeons interested in improving the treatment of hand trauma, that to the individual's future his phalangeal fracture may be more important than a fractured femur, tibia or humerus, and deserves as much or more time, thought, and experienced care as trauma to the major long bones.

Dr. Brooks has carried you skillfully through the fundamental elements of treatment, reduction, immobilization, and restoration of function of phalangeal fractures. I heartily endorse these principles and technics and am convinced that their skillful application will enhance the end results of fractured fingers in any practice.

Forhistal, A New Type of Low-Dosage Antihistamine, For the E.N.T. Patient

• Report of the use of a new antihistaminic drug administered to one hundred patients in an outpatient clinic.

WERNER J. HAGEN, M. D.
Houma

THE specialist in ear, nose and throat often is called upon to treat certain types of allergic patients because these systems are frequently involved symptomatically. Likewise, intercurrent upper respiratory infections in allergic patients may produce more frequent E.N.T. complications such as tubotympanitis, serous otitis media, requiring the specialist's attention. Since the allergic problems in such cases often are those that respond well to symptomatic antihistamine therapy, familiarity with the drugs utilized is a necessity. Needless to say, proper referral must be made for the patient whose allergy requires more definitive treatment.

The significance and acceptability of the antihistamines in allergic disorders need no elucidation on my part; the variety used in practice attests to that. Recently, however, there have been developed several highly potent, low-dosage antihistimines which have begun to replace the standard products of the past.

The most recent addition to these new antihistamines belongs to a new series of chemical compounds, the indenes, and has demonstrated high potency in low dosage with few of the usual antihistamine side effects. This drug has the generic name dimethpyrindene* and is designated as:

2-(1-(2-(2-dimethylaminoethyl)-3-indenyl)-ethyl)-pyridene maleate.

Details of the pharmacologic and other laboratory studies need not be cited here since they have been noted in previous publications.[1, 2] Suffice it to say that dimethpyrindene in laboratory animals has shown excellent antihistamine activity, more prolonged than that of certain other widely used drugs used in treating allergic patients, and a very low order of toxicity.

Clinical studies by a large number of physicians, several of whom have published their findings,[3-8] have demonstrated the effectiveness of dimethpyrindene in a wide variety of allergic patients from infancy to old age. Seasonal allergies and pruritus have responded particularly well; rapid absorption of the orally administered drug permits early onset of action and produces maximal effects within an hour. Analysis of the cumulative clinical reports indicates less sedative action than with several other antihistamines; other side effects are practically negligible. Duration of symptomatic control is about three to four hours with the 1 mg. tablets of dimethpyrindene. The 2.5 mg. Lontabs® have an equally rapid onset of action and provide relief for about eight hours.

Clinical Study

One hundred patients from the outpatient clinic of our ear, nose and throat hospital were selected as subjects for evaluation of dimethpyrindene. Ages ranged from 3 to 60 years and both sexes were included. Dosage ranged from 0.5 mg. t.i.d. to 1.0. q.i.d., being adjusted individually according to severity of symptoms and response.

Diagnostic categories and responses to treatment are summarized in Table 1. It will be noted that a few cases (categories C and D) were treated despite absence of E.N.T. symptoms; this was done simply because they were old patients of our clinic.

* Forhistal® maleate, Ciba.

TABLE 1
EFFECTS OF DIMETHPYRINDENE IN 100 PATIENTS

Diagnoses	Number of Pts.	Symptomatic Responses			
		Very Good	Good	Fair	Poor
A. Perennial Rhinitis	14	8	4	1	1
B. Seasonal Rhinitis	35	22	12	0	1
C. Pruritis, probably allergic	3	2	0	0	1
D. Acute Urticaria with severe pruritis	3	3	0	0	0
E. U.R.I., uncomplicated	15	10	3	1	1
F. U.R.I. with E.N.T. complications	23	17	3	1	2
G. Tubotympanitis, serous otitis media	7	3	2	0	2
TOTALS	100	65	24	3	8

It should be noted that if E.N.T. complications required additional therapeutic measures, simple surgery (i.e. myringotomy) and the appropriate antibiotic were utilized along with dimethpyrindene administration.

Our results as to optimum responses of seasonal rhinitis and pruritis were substantially in agreement with those of other investigators. Perennial rhinitis cases were somewhat less responsive, but results still compared well with the use of other antihistamines.

In acute coryza the best drug responses were found in those cases having eosinophiles in the nasal smear.

Of most immediate importance in our special field, we found dimethpyrindene valuable as part of the management of upper respiratory infections with complications such as tubotympanitis, low-grade sinusitis and serous otitis media. This type of otitis, especially, appeared to resolve more rapidly with the use of this drug.

Side effects were minimal and occurred altogether in less than 10 per cent of patients. Slight drowsiness was the most frequently reported complaint, but it did not necessitate discontinuance of the drug in any case.

About a third of the patients who were questioned as to duration of symptomatic relief stated that dimethpyrindene seemed better than drugs used previously.

Conclusion

Dimethpyrindene has excellent specific applicability to problems commonly presented to the ear, nose and throat specialist, and it is our belief that it is well worth further clinical use. The effectiveness and freedom from severe side effects of this member of a new chemical series introduce a promising new antihistamine development to the medical profession.

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Proceedings Southern Society of Anatomists First Annual Meeting, Tulane University Nov. 3-4, 1961

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1. Studies on the migration of the epithelium on the external surface of the membrana tympani. **Peter W.R.M. Alberti,** Department of Anatomy, Emory University.

2. A neurophysiological technique for studying a small number of fibers that join a larger nerve. **F. Eugene Doenges,** University of Missouri School of Medicine.

3. Anatomical problems in the transplantation of the canine liver. **John E. Healy, Jr., M. D.,** The University of Texas Post-Graduate School of Medicine.

4. A method for the microscopic study of the living lung in the closed thorax. **Vernon E. Krahl, William E. De Alva, W. Gerald Rainer,** and **Giles E. Filley,** Cardiopulmonary Laboratory, Univ. of Colorado Medical Center.

5. Conversion of Collagen and Reticulum Fibers into "Elastic Fibers". **G. Kuhns, Holde Puchtler,** and **Faye Sweat,** Department of Pathology, Medical College of Georgia.

6. Histochemical and chromatographic studies on the plasmalogens of infarcted skeletal muscle of the dog. **Richard M. Landry** and **Victor J. Ferrans,** Tulane University.

7. An electromyographic study of selected muscles acting on the wrist. **Gordon B. McFarland, Jr., Hal T. Weathersby,** and **Ursula L. Krusen,** Departments of Physical Medicine and Rehabilitation and Anatomy, University of Texas Southwestern Medical School.

8. Hypertension induced by means of X-rays: with histochemical studies. **Robert McKelvey,** Department of Anatomy, Emory University.

9. Hypoxia-hypercapnia, cooling and asphyxia in kittens. **Faith S. Miller** and **James A. Miller,** Department of Anatomy, Tulane University.

10. Recovery of regenerative ability in limbs of Triturus after x-irradiation when normal epidermis is added. **S. Meryl Rose** and **Florence C. Rose,** Department of Anatomy, Tulane University.

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1. Studies on the migration of the epithelium on the external surface of the membrana tympani. Peter W.R.M. Alberti, Department of Anatomy, Emory University.

The fate of the external layer of the membrana tympani is assuming great clinical importance. This has been studied by painting the external layer of the drum with dye in human subjects. It is suggested that in a large portion of cases the epithelium migrates centrifugally from the region of the umbo, across the drum on to the walls of the bony external canal, eventually being shed at the bony-cartilaginous border, and eliminated with cerumen.

2. A neurophysiological technique for studying a small number of fibers that join a larger nerve. F. Eugene Doenges, University of Missouri School of Medicine.

The technique is based on those of Ritchie ('57a, '57b) but differs in that nicotine is used; and also, in that after the large nerve complexes are decreased in size by activating impulses in the small number of fibers joining the large nerve, they are then increased in size by blocking any activated and "spontaneous" impulses in the small component. Nicotine is used because of its dual action to activate and then block conduction in nerve

fibers, and because of its selective action to block synaptic junctions in a ganglion more readily than fibers passing through the ganglion.

The procedure is to record the potential complexes of the large nerve for a time before and after application of nicotine, with or without electrical stimulation, to the small component or to the ganglion if there is one in the course of the component. The alteration of activity in the small component will be revealed in the alteration of the recorded complexes of the large nerve.

In many peripheral nerves which have a small component of fibers from a ganglion, this technique will provide an approximation of what percentage of the fibers in the large component are from the small component and in certain cases may provide information as to nature of the fibers in the small component.

3. **Anatomical problems in the transplantation of the canine liver.**[1] **John E. Healey, Jr., M. D.,** The University of Texas Post-Graduate School of Medicine.

Whole organ transplantation in the experimental animal is an extremely difficult procedure because of the numerous vascular anastomoses which need to be performed. In liver transplantation, the inferior vena cava both above and below that organ, as well as the portal vein and the hepatic artery, must be re-approximated by end-to-end anastomosis in order to restore hepatic circulation. With a trained surgical team, this phase of the procedure requires approximately one hour to complete. The anoxic condition of the liver during this time almost invariably results in the production of a state of irreversible shock. We have devised a means of re-establishing hepatic circulation within a period of seven minutes by means of a specially designed vascular clamp (anastomat). The clamps are easily applied, permit a leisurely repair, and can be removed from the vessel with ease following the repair.

[1] Aided in part by an American Cancer Society Institutional Grant.

Other anatomical problems including reestablishment of biliary flow, the ligation of the phrenic veins, and the maintenance of portal and caval blood flow during the transplantation procedure will be discussed.

4. **A method for the microscopic study of the living lung in the closed thorax.**[2] **Vernon E. Krahl,**[3] **William E. De Alva, W. Gerald Rainer,** and **Giles F. Filley.** Cardiopulmonary Laboratory, U. of Colorado Medical Center.

The most accurate histophysiologic information is gained by observing living tissues. The complete dependence of the lung for its form, dimensions and normal function upon (a), a closed thorax and (b), the mechanical forces of the moving diaphragm and thoracic walls requires that this organ be studied in the living animal and in the closed thorax. The following method provides for the microscopic study of the lung under these conditions.

A permanent window of glass and stainless steel is installed in the rabbit's lateral thoracic wall. The lung surface may be observed by incident light supplied through the Ultropak objective. For the study of deeper structures by transillumination, a Teflon port is placed in the lateral abdominal wall about three inches caudal to the thoracic window. During observation of the lung the port is fitted with a vinyl sleeve. A quartz rod (Knisely apparatus) is passed into the peritoneal cavity within the sleeve until its tip touches the diaphragm opposite the thoracic window. The translucent diaphragm readily permits light conducted by the rod to transilluminate the lung. Heat at the tip of the rod is dissipated by water flowing through the vinyl sleeve.

Observations may be made immediately following installation of the windows and at intervals for one month or longer. The

[2] Supported in part by Training Grant No. HTS 5450 and HTS 5450(S1) from the National Heart Institute of the Public Health Service.

[3] Permanent addless: Department of Anatomy, University of Maryland School of Medicine, Baltimore, Maryland.

method allows the study of normal alveolar dimensions and pulmonary circulation, but also may be used to study responses of lung tissue to air pollutants, activity of phagocytes and the development of various pulmonary diseases.

5. **Conversion of collagen and reticulum fibers into "elastic fibers".**[4] **G. Kuhns, Holde Puchtler,** and **Faye Sweat,** Department of Pathology, Medical College of Georgia.

Acetylation, sulfation or phosphorylation induce binding of resorcin-fuchsin by basement membranes, reticulum fibers, collagen, and other tissue structures containing polysaccharides. Extraction procedures indicate binding of resorcin-fuchsin by non-ionic bonds, presumably hydrogen bonds between phenolic hydroxyl groups of the dye and ester groups in the tissue.

Electron microscopic studies by Hall have shown that collagen can be converted into elastica-like material. It seemed interesting to investigate whether such pretreated fibers also acquire the staining properties of elastic fibers. Sections of human kidneys, fixed in absolute alcohol or Carnoy's fluid and embedded in paraffin, were treated with hot distilled water, acetic, trichloracetic, formic, oxalic, hydrochloric, or nitric acid, borate buffer, 10% $CaCl_2$ or 0.1 N NaOH, and stained with resorcin-fuchsin. Collagen and reticulum fibers were intensely colored, but basement membranes remained unstained.

The intense staining of collagen and reticulum fibers with resorcin-fuchsin indicates that binding of this dye does not convey any information concerning the protein moiety of tissue structures, whether it is collagen or elastin, but apparently demonstrates compounds associated with proteins, presumably polysaccharide esters. Thus, resorcin-fuchsin can not be considered specific for elastic fibers in the chemical sense of the term.

6. **Histochemical and chromatographic studies on the plasmalogens of infarcted skeletal muscle of the dog.**[5] **Richard M. Landry**[6] and **Victor J. Ferrans,**[7] Tulane University.

Experimental infarction of skeletal muscle was produced by means of injection of ion exchange resin beads (Dowex 50X-16, hydrogen form, 100 mesh) into the femoral artery of dogs. The contralateral leg was used as a control throughout.

Fresh-frozen cryostat sections of the infarcted areas of muscle showed a marked decrease in the concentration of plasmalogens as determined by plasmal reaction, along with an increase in the concentration of neutral lipids as evidenced by staining with Sudan Black B, Phosphine and Nile Blue.

Paper chromatographic analysis of the lipids extracted from the infarcted muscle confirmed these findings and in addition showed the presence of a new plasmalogen with chromatographic properties similar to those of the plasmalogen previously found by Hack and Ferrans (Circ. Res., VIII, 738, 1960) in infarcted heart muscle of the dog.

The significance of these findings will be discussed.

7. **An electromyographic study of selected muscles acting on the wrist. Gordon B. McFarland, Jr., Hal T. Weathersby,** and **Ursula L. Krusen,** Departments of Physical Medicine and Rehabilitation and Anatomy, University of Texas Southwestern Medical School.

The kinesiology of 10 muscles acting on the wrist has been studied by means of multichannel electromyography. An electrogoniometer was used to record the position of the wrist simultaneously with the action potentials of the muscles producing the motion. Eighteen normal subjects were used in this study.

The principal radial abductors were the extensor carpi radialis longus and brevis, abductor pollicis longus, flexor carpi ra-

[4] Supported by USPHS research grant #RG-7303 and Medical College of Georgia Professional Research Fund Grant #51-79.

[5] This investigation has been supported by grant HTS 5133 from the National Institutes of Health, United States Public Health Service.

[6] Fellow of the Cardiovascular Research Graduate Training Program, Tulane University.

[7] National Heart Institute Research Fellow in Cardiology, Department of Medicine, Tulane University School of Medicine.

dialis and extensor pollicis brevis. The amplitude of spikes from the extensor carpi ulnaris was usually much greater than that of the flexor carpi ulnaris in ulnar deviation. The extensor digitorum and the flexor digitorum superficialis showed activity at the extremes of both radial and ulnar deviation.

All posterior antebrachial muscles crossing the wrist joint were involved in simple extension of the wrist, with the extensor carpi radialis brevis and extensor carpi ulnaris being most active. However, the pattern of activity was changed when both the wrist and metacarpophalangeal joints were extended simultaneously, in which actions a reciprocal relationship existed in most subjects between the extensor carpi radialis brevis and the extensor digitorum. Flexor antagonist activity was uniformly present when the wrist was extended and the metacarpophalangeal joints were hyperextended.

8. **Hypertension induced by means of X-rays: With histochemical studies.**[8] **Robert McKelvey,** Department of Anatomy, Emory University.

Hypertension was induced by x-ray irradiation directed to both kidneys of unoperated rats. The irradiation was administered in a single dose of 1200r to one group and 3000r in fifteen fractions to a second group. Blood pressures were determined at intervals for insulted and control animals.

Some histochemical studies were made on sections of kidneys. The enzymes investigated included: succinic dehydrogenase, glucose-6-dehydrogenase, glutamic dehydrogenase, and glucose-6-phosphatase.

9. **Hypoxia-hypercapnia, cooling, and asphyxia in kittens.**[9] **Faith S. Miller** and **James A. Miller,** Department of Anatomy, Tulane University.

Studies on neonatal puppies and guinea pigs have established the principle that pretreatment with hypoxia-hypercapnia protects mammals from some of the deleterious effects of hypothermia, thereby increasing the protection against asphyxia conferred by reduction in body temperature. Confirmatory results have been obtained for neonatal cats. The survival time of kittens when asphyxiated after cooling to 15° body temperature without hypoxia-hypercapnia was approximately 10 times as long as that of normothermic littermates. However, when hypoxia and hypercapnia were induced during cooling kittens lived nearly twice as long as littermates which had been cooled rapidly while breathing air. Results were similar whether the hypoxia and hypercapnia were produced by rapid lowering of temperature in icewater (5 minutes) while the animals breathed 10% O_2 + 5% CO_2 or by slow cooling (45 minutes) while the animals rebreathed air in a closed vessel packed in ice.

10. **Recovery of regenerative ability in limbs of Triturus after x-irradiation when normal epidermis is added. S. Meryl Rose** and **Florence C. Rose,** Department of Anatomy, Tulane University.

Limbs of adult *Triturus* were x-rayed with a dosage of 2000r. This prevents regeneration. Radioautographs of limbs x-rayed and treated with tritiated thymidine indicate that a dose of 2000r does not prevent incorporation of the thymidine into DNA and mitosis does not decrease in the x-rayed epidermis, but internal changes after amputation associated with regeneration do not occur or occur very slowly. The usual loss of internal structure and concurrent elaboration of a blastema fails.

If limbs are amputated and the x-rayed stumps provided with a normal epidermis limb regeneration ensues. In contrast to the totally x-rayed limbs, x-rayed limbs provided with normal limb epidermis produce a blastema and all missing parts regenerate. Preparations stained for nerves indicate that the normal dose association of nerves and wound epidermis is lacking after x-irradiation and amputation. When x-rayed limbs are provided with a normal epidermis, nerves penetrate the epidermis in great numbers.

[8] Supported by a Grant from the Life Insurance Medical Research Fund.

[9] Supported by N.I.H. Grant #B2279.

The Journal of the Louisiana State Medical Society

Established 1844

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News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor, 1430 Tulane Ave., New Orleans, La.

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Editorial

Diabetic Neuropathy

Diabetes mellitus as a clinical entity has been recognized for a greater time than most other diseases. It has been described since the time of Moses. Apparently it is increasing in frequency at the present time. There are now about one million five hundred thousand known cases of diabetes in the United States. An equal number, it is estimated, exists in the population whose condition is unknown to themselves and to their physicians. If those individuals were included whose only evidence of diabetes at the present time is a positive glucose tolerance test the numbers in both categories would be even greater.

Diabetic neuropathy has been recognized since 1798. But its neurological complications have been a serious source of concern principally in the last seventy-five years. Even now, accurate definition of what constitutes diabetic neuropathy is somewhat confused and the prevalence of the condition is affected in the same way. The percentage of individuals having diabetes mellitus and giving evidence of neuropathy is variously reported as ranging from 0 to almost 100 per cent. It is probable that approximately one-half of diabetics will show evidence of neuropathy, if carefully studied, at some time in their course.

Its manifestations and recognition therefore become a matter of grave importance in all fields of medicine, particularly among those who see the most diabetics, namely, the general practitioner and the internist. The manifestations of diabetic neuropathy may be grouped as sensory, motor—including myopathy, and autonomic. The most frequent disturbance in diabetic sensory neuropathy is pain in the legs (often nocturnal), muscle tenderness, loss of Achilles reflexes, and loss of vibratory sensation of feet and ankles. The motor disorders have included isolated nerve palsies of the oculomotor, abducens, and facial nerve; and certain au-

thorities have described a purely motor diabetic syndrome among the manifestations of which are weakness and wasting of muscles. Syndromes of diabetic neuropathy in the autonomic nervous system include: paroxysmal nocturnal diarrhea, impotence, bladder paralysis, loss of sweating, postural hypotension, postural tachycardia, neuropathic joints, and perforating ulcers.

The biochemical processes by which the degeneration of nerve pathways is brought about is not clear. The causes of the nerve degenerations have been considered under vitamin deficiency, hyperglycemia and intermediate metabolic disturbance, and ischemia from arterial changes caused by or developing from the diabetes. It is the opinion of most investigators that the problem is not one of vitamin deficiency, and if such exists, it is a concomitant disease and may react upon the neuritis, clouding the picture but not causing it.

Ischemia from arterial changes is in the same category. The actual cause of damage to the nerve tissue is probably an intermediate metabolic disturbance associated with, or due to the hyperglycemia—probably the former. It has been suggested that deviations in the pyruvate metabolism are related to the degenerative process of diabetic neuropathy.

Etiological factors related to the circumstances in the patient with diabetes, who is going to develop diabetic neuropathy, have been the subject of considerable discussion. A typical summary has been: "peripheral neuritis and diabetes is a common complication of neglected diabetes over long periods." An opposing view [1] is that neuropathy may occur during good control and there may be simultaneous onset of neuropathy and the symptoms of uncontrolled glycosuria. Neuropathy may be unrelated to the duration or severity of the diabetes. Neuropathy may be the initial clinical manifestation of diabetes unattended by symptoms of hyperglycemia and glycosuria. Paradoxic precipitation of neuropathy following the institution of good control by diet, insulin, tolbutamide has been observed.

Neuropathy may follow stress situations. In these instances, a relatively constant latent period exists.[1] Diabetic neuropathy in this view should be regarded as an essential integral feature of the syndrome of diabetes mellitus, rather than a complication of the disease. Such a review has also been stated in regard to retinopathy by other authorities.

The diagnosis of diabetic neuropathy, although not difficult, is generally one of the least precise clinical diagnoses. The disturbing factor is that the newly discovered diabetic or the patient with diabetes which is not properly controlled is subject to many symptoms of a sensory nature which may vary from minor hypesthesias to a syndrome of an acute surgical abdomen, but without demonstrable nerve lesions. Subsequently, the diabetic may develop a peripheral diabetic neuropathy resembling in many respects those associated with other types.

Manifestations of this include: muscular cramps and aching, muscular weakness—especially of the legs, possibly a flaccid paralysis or bilateral wrist-drop or foot-drop, severe shooting pains or hypesthesias, areas of anesthesia or unsteadiness of gait, and in all these disturbances the lower extremities were more severely affected than the upper. These symptoms would be intensified following acute infections and lapses in the proper care of diabetic management.

The treatment of this condition is the proper and complete treatment of the diabetes itself. In addition to this, to provide for the comfort and the disabilities of the patient, the special manifestations should be met in conditions such as pain, hypesthesia, perforating ulcers, neurotrophic joint destruction, paralyses, and depressive emotional reactions to the associated disorders.

Accordingly, the physician in dealing with diabetes is continually called upon to take into consideration its protean manifestations, including degenerative lesions in the nervous system.

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[1] Ellenberg, Max: Diabetic Neuropathy: A consideration of factors in onset, Ann. Int. Med. 52:1067 (May) 1960.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

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LIAISON WITH LOUISIANA HOSPITAL ASSOCIATION AND NURSES ASSOCIATION
Dr. Philip H. Jones, New Orleans

LIAISON WITH LOUISIANA NURSES ASSOCIATION
Dr. C. Walter Mattingly, New Orleans

LIAISON WITH LOUISIANA STATE BAR ASSOCIATION
Dr. Daniel M. Kingsley, Alexandria

LIAISON—PUBLIC POLICY AND LEGISLATION AND CONGRESSIONAL MATTERS
Dr. Arthur D. Long, Baton Rouge
Dr. B. O. Morrison, New Orleans
Dr. C. Grenes Cole, New Orleans

LOUISIANA ORGANIZATIONS FOR STATE LEGISLATION
Dr. Daniel J. Fourrier, Baton Rouge

MATERNAL WELFARE
Dr. R. G. Masterson, Alexandria

MEDIATION
Dr. Nicholas J. Chetta, New Orleans

MEDICAL DEFENSE
Dr. C. B. Erickson, Shreveport

MEDICAL EDUCATION
Dr. Edwin H. Lawson, New Orleans

MEDICAL AND HOSPITAL SERVICES IN RE INSURANCE CONTRACTS
Dr. Ralph M. Hartwell, New Orleans

MEDICAL TESTIMONY
Dr. Henson S. Coon, Monroe

MENTAL HEALTH
Dr. F. W. Brewer, New Orleans

NATIONAL EMERGENCY MEDICAL SERVICE
Dr. Moss M. Bannerman, Baton Rouge

NEUROPSYCHIATRIC SERVICE AT CHARITY HOSPITALS
Dr. P. H. Jones, New Orleans

NOMINATIONS (Committees)
Dr. J. Kelly Stone, New Orleans

PUBLIC HEALTH OF THE STATE OF LOUISIANA
Dr. W. J. Rein, New Orleans

PUBLIC POLICY AND LEGISLATION
Dr. Jos. A. Sabatier, Baton Rouge

REVISION OF CHARTER, CONSTITUTION AND BY-LAWS
Dr. A. V. Friedrichs, New Orleans

RURAL AND URBAN HEALTH
Dr. Guy R. Jones, Lockport

STATE DEPARTMENT OF PUBLIC WELFARE
Dr. H. H. Hardy, Jr., Alexandria

STATE GOVERNMENT HEALTH SERVICES
Dr. Walter Moss, Lake Charles

STATE HOSPITAL POLICIES AND MEDICAL INDIGENCY
Dr. W. Robyn Hardy, New Orleans

TETANUS PROTECTION
Dr. W. P. Gardiner, New Orleans

WOMAN'S AUXILIARY
Dr. C. Grenes Cole, New Orleans

MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Ascension	Third Tuesday of every month	
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Jefferson	Third Thursday of every month	
Lafayette	Second Tuesday of every month	Lafayette
Morehouse	Third Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Tangipahoa	Second and fourth Thursdays of every month	Independence
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

SURGICAL ASSOCIATION OF LOUISIANA

The Surgical Association of Louisiana will hold its annual meeting on Sunday, November 12, 1961, headquarters at the Sheraton-Charles Hotel, New Orleans.

A panel discussion on Chemotherapy of Tumors will begin at 9:30 a.m., with Dr. Ambrose H. Storck of New Orleans as Moderator. Beginning at 1:00 p.m., there will be a panel discussion on Hiatal Hernia with Dr. Hugh Ilgenfritz of Shreveport as Moderator.

Out-of-State speakers are Edward H. Ellison, M. D., Professor and Chairman, Division of Surgery, Marquette University School of Medicine, Milwaukee, and Carl A. Moyer, M. D. Bixby Professor of Surgery and Head of the Department, Washington University School of Medicine, St. Louis.

AMA CLINICAL MEETING SET NOV. 26-30 AT DENVER

The 15th annual clinical meeting of the American Medical Association will be held November 26-30 at Denver, with a program geared to basic problems of medicine faced by physicians in their practice.

An outstanding scientific program, with emphasis on new research developments, has been planned under the direction of Samuel P. Newman, M. D., Denver, chairman of the AMA'S Council on Scientific Assembly.

Some highlights will include sessions and papers on such important areas of medicine as genes and chromosomes, electronics and computers in medicine, space medicine, medical aspects of American habits, new developments in virology, treatment of radiation injuries, new findings in chemotherapy for cancer and latest data in the field of antibodies and antigens, Dr. Newman said.

With more and more nuclear reactors coming into use all over the nation, many practicing physicians soon may begin to face the problem of treating injuries from radiation accidents, the chairman said.

A section of internationally known experts in the treatment of radiation injuries will offer three major papers in this important new area of medical care. Chairman will be Marshall Brucer, M. D., chairman of the medical division, Oak Ridge Institute of Nuclear Studies, Oak Ridge, Tenn.

The radiation experts will discuss such topics as "Potential and Probable Sources of Radiation Accidents," "Diagnosis and Pathology of Radiation Injury" and "Treatment and Prognosis of Radiation Injury." Participants will include researchers from Los Alamos and Oak Ridge, the Office of the Surgeon General and the University of Chicago.

The age of advancing physical science also offers new findings to medical science: the use of electronics and computers in medicine. Chairman of this section at the Denver meeting will be A. H. Schwichtenberg, M. D., head of the department of aero-space medicine, Lovelace Foundation for Medical Education and Research, Albuquerque, N. M.

Computer systems for recording medical data to aid the physician in his diagnosis and prognosis will be discussed. Topics will include "The Future of Electronics in Medicine," "Microelectronics and New Concepts of Bioinstrumentation," "A System for Medical Data Recording," and "Biological Computers."

The virus, one of the most complex problems facing the clinician, will be the subject of a series of papers by outstanding specialists. Jonas E. Salk, M. D., Pittsburgh, originator of the killed virus polio vaccine, will give a paper on "Immunization Against Virus Diseases." Other topics will include "The Nature of the Virus and Its Cellular Reaction," "Smallpox Vaccination Complications," "Virus Hepatitis" and "Identification of Viruses."

"We are confident that the 15th annual clini-

cal meeting will offer one of the most interesting and informative programs ever presented at the winter session," Dr. Newman said.

"The program is designed to assist the physician in his practice. The latest findings in many areas of medicine will be presented by men who are top specialists in their fields. The meeting will be of great value to the clinician in advancing his knowledge."

1961 CONVENTION, LOUISIANA ACADEMY OF GENERAL PRACTICE

Dr. Francis I. Nicolle, Secretary of the Louisiana Academy of General Practice and 1961 Convention Chairman, has announced that the Academy will hold its fifteenth annual scientific assembly at the Hotel Monteleone in New Orleans on October 17-19, 1961. This program is accepted for eleven hours Category I credit by the American Academy of General Practice. Registration fee will be $5.00.

Dr. Rafael C. Sanchez of New Orleans, Speaker, will open the sessions of the Congress of Delegates on the morning of October 17. Dr. John G. McClure of Welsh is Vice-speaker of the Congress.

The Scientific Assembly will begin on the afternoon of October 17 with Dr. Ernest B. Flake as presiding officer. Welcoming addresses will be made by the Honorable Victor H. Schiro, Mayor of New Orleans; Dr. J. Theo Brierre, President of the Orleans Parish Medical Society, and Dr. Farrell R. Nicholson, President of the Jefferson Parish Medical Society. Dr. Nicholle will make the response for the Academy.

Following is a list of guest speakers and their topics: Recognition and Management by the General Practitioner, by Dr. Wm. G. Crook, Jackson, Tenn.; Pelvic Pain, What It Is and What To Do About It, by Dr. Wm. F. Guerriero, Dallas, Tex.; Allergy in Children, Part II, by Dr. Wm. G. Crook, Jackson, Tenn.; Clinicopathologic Conference, Dr. Andrew S. Ranier, Lake Charles, La.; Carcinoma of the Cervix, the Endometrium and the Ovary, by Dr. Wm. F. Guerriero, Dallas, Tex.; The Peculiar Looking Child, by Dr. Sydney S. Gellis, Boston, Mass.; Treatment of Acute Hand Injuries, by Dr. Daniel C. Riordan, New Orleans; The Use of Gamma Globulin in Infancy and Childhood, by Dr. Sydney S. Gellis, Boston, Mass.; Treatment of Fractures of the Hand, by Dr. Daniel C. Riordan, New Orleans; Some Aspects of Estate Planning, by Mr. Arthur J. Riggs, Dallas, Tex. and A Systematic Approach to the Use of Laboratory Determinations for Diagnosis, by Dr. Andrew S. Ranier, Lake Charles, La.

The Woman's Auxiliary will hold its annual business session and installation of officers during the program. Mrs. E. A. Bertucci, Jr., assisted by Mrs. Pierre A. Donaldson, has arranged a full program of social activities for the ladies.

Dr. Boyd M. Woodard of Lake Charles is President-Elect.

SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY SOUTHERN MEDICAL ASSOCIATION

The Section on Ophthalmology and Otolaryngology of the Southern Medical Association, will meet in Dallas, Texas, on November 6-9, 1961.

The program will feature Television surgical clinics on Glaucoma and Laryngectomy. Also, papers and symposiums on Corneal Diseases; Contact Lens; Retinal Detachment; Tympanoplasty; Otitis Media; Tonsillar Techniques, etcetera.

Dr. Trygve Gundersen of Boston, Massachusetts, will be the guest speaker.

For additional information please contact the secretary, Dr. Albert C. Esposito, First Huntington National Bank Building, Huntington, West Virginia.

SECOND POSTGRADUATE SEMINAR INTERNATIONAL MEDICAL-LEGAL SOCIETY

The International Medical-Legal Society announces its second postgraduate seminar to be held in Honolulu February 19-23, 1962.

The goal is to determine the functions of the doctor, the lawyer, and the client in legal cases, and then formulate ethics and rules for doctor-lawyer relations.

This society was founded to promote the interprofessional relations between Doctors and Attorneys. A panel of physicians and attorneys has been selected to equip the attorneys with more adequate medical knowledge, and the physicians with a comprehensive review of the law of medical practice. This is a joint society for both Attorneys and Physicians who deal in medical legal cases or are subject to malpractice.

A five-day meeting has been prepared. Physicians subject to malpractice as well as medical legal work and attorneys involved in similar suits will find the meeting informative and of great value. Speakers of national recognition from both the Medical and Legal professions will be on the program.

The Where-To-Go Travel Service of Seattle has been selected as the sole travel agency to prepare an organized package tour making all travel arrangements for the meeting.

Reservations will be limited as this is the peak travel season for Hawaii. Those interested may contact Wm. P. Hauser, M. D., Executive Secretary, 1206 South 11th, Tacoma, Washington, for further information.

BOOK REVIEWS

Complications in Surgery and Their Management; by Curtis P. Artz, M. D. and James D. Hardy, M. D., Saunders, 1960, 1075 p. $23.00.

With the aid of 69 able contributors, Dr. Artz and Dr. Hardy have compiled a comprehensive volume on the complications of surgery and their management. As stated by the editors in their preface of the book, it was their desire to provide a single text on surgical hazards to be used along with other texts of of surgery, surgical technique, surgical physiology, and surgical pathology. In this respect, they have provided an unique volume which accomplishes their goal admirably.

The format of the book consists of an initial twenty chapters devoted to complications encountered in all types of surgery. General topics such as complications of parenteral fluid therapy, postoperative pulmonary complications, diabetes and nutritional complications in surgical patients, and thromboembolic diseases are discussed. One chapter in this portion of the book discusses miscellaneous complications of tracheotomy, paracentesis, angiography, and drains. The remaining 37 chapters include discussions of complications encountered in specific operations such as: mitral comissurotomy, esophageal and diaphragmatic surgery, surgery of the peripheral arteries, gastric resection, biliary tract surgery, and many others. There are two chapters dedicated to problems of surgery of the aged and pediatric surgery. Complications arising in gynecologic, urologic, and orthopedic procedures are included in three chapters devoted to these surgical specialties. The final chapter of the book considers the pitfalls of burn therapy.

The authors of the text are an impressive group of outstanding authorities in the field of their contribution. Each chapter is prefaced with a brief biographical sketch of the author which is an interesting sidelight that should be used more frequently in medical texts. The book is a valuable reference for all surgeons and particularly for residents and interns. It should be used not only as a reference once a complication arises but also as a preoperative reference to anticipate complications and study methods of preventing them.

W. L. CHAPMAN, M. D.

Recent Advances in Tropical Medicine; by Sir Neil Hamilton Fairley, A. W. Woodruff and John H. Walters, Little Brown and Company, Boston, Third Edition 1961. 480 p. $11.00.

This is a completely new volume since the Second Edition was published thirty-two years ago. It therefore covers a period during which more advances have been made in therapy and etiology of tropical diseases than any comparable period.

These authors have emphasized the clinical aspect and therapy, although there are many short sections on epidemiology, pathology and control. The book will appeal more to the practicing physician rather than the public health officials.

It is an excellent book that through the experience of the authors links the present knowledge with the past. They cover most of the important protozoa, helminthic, bacterial, spirochaetal diseases, short term fevers, venereal diseases, rickettsial diseases, nutritional diseases, and anemia including the heriditary hemoglobinopathies, Kuru and some diseases of uncertain origin. Their index is excellent.

E. A. CLEVE, M. D.

The Modern Treatment of Asthma, with Special Reference to Gold Therapy; by L. Banszky, Williams & Wilkins, 1959, 2d ed., pp. 175. Price $5.00.

This provocative book is based on a concept almost certainly incorrect, namely, that the root condition of asthma is due to tuberculinization of the organism (p. 39). The sequel to this, that patients can be helped with gold injections is, however, not necessarily wrong. It is known that a variety of agents which help rheumatoid arthritis also help bronchial asthma; among them may be mentioned the steroid drugs and nitrogen mustard. Gold therapy is of distinct value in the treatment of rheumatoid arthritis, and by extrapolation might also be useful in the treatment of bronchial asthma. In view of his results of 80 to 90 percent success rate, perhaps this method of treatment should be tried more extensively.

VINCENT DERBES, M. D.

W. B. SAUNDERS COMPANY features the following recent books in their full page advertisement appearing elsewhere in this issue:

DRIPPS, ECKENHOFF AND VANDAM—INTRODUCTION TO ANESTHESIA

An ideal basic guide to the understanding and safe administration of anesthesia

CORDAY AND IRVING — DISTURBANCES OF HEART RATE, RHYTHM AND CONDUCTION

Covers management of **all** the cardiac arrhythmias and conduction defects

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PUBLICATIONS RECEIVED

(Certain ones of these will be selected for review)

Doubleday & Co., N. Y.: Traitor Within Our Suicide Problem, by Edward Robb Ellis and George N. Allen.

The C. V. Mosby Co., St. Louis: Pathology, by W. A. D. Anderson, M. D. (4th edit.).

W. B. Saunders Co., Phila.: Respiration in Health and Disease, by R. M. Cherniack, M. D., and L. Cherniack, M. D.; The Cervix Uteri and Its Diseases, by C. Frederic Fluhmann, M. D.